The Journal of Pediatrics

VOL. 5 DECEMBER, 1934 NO. 6

Original Communications

A STUDY OF FORTY CASES OF PERSISTENT PYURIA

PEARL SUMMERFELDT, M.B., AND ALAN BROWN, M.D.
TORONTO, ONTARIO

IN THIS present paper are the results of a study of forty cases of persistent pyuria. The investigation has included a routine examination of the urinary tract by means of intravenous pyelography, cystoscopic examination and retrograde pyelograms. Cystograms were made when indicated. Besides these there was a bacteriologic examination of the urine,[1] nonprotein nitrogen and creatinine determinations of the blood, and an estimation of the blood pressure.

In this series there were thirty-five females and five males. Of the forty patients there were nine under two years of age, 14 patients between two and five years of age, and seventeen patients over five years of age. The youngest was a two-and-one-half-week-old male infant, who died of a severe infection of the left kidney following the removal of the right kidney for a similar infection. There were three other deaths in this series, one being due to a septicemia which had no relationship to the kidney infection, and the other two died of a chronic interstitial nephritis secondary to their persistent pyuria.

Clinically persistent pyuria is readily recognized. In twenty-seven cases a diagnosis was made on the clinical history before urinalysis was done. In these there were complaints of a long duration which were mostly referable to the urinary tract, such as day and night frequency, abdominal pain, urgency, and cloudy urine. An elevated temperature was not always present. In the remaining thirteen cases not recognized on admission as pyuria before the urinalyses were done, there were

From the Subdepartment of Paediatrics, University of Toronto, and the Hospital for Sick Children.

various diagnoses made, such as malnutrition, nasopharyngitis, digestive upset, meningitis, adenitis, cystitis, and renal calculus.

Cases of persistent pyuria admitted within the first four weeks of the onset of the illness have to be diagnosed from acute pyuria. This is either done by following the case over a prolonged period of time or making an intravenous pyelogram.

Although as a rule the majority of cases showing persistent pus in the urine are due to an interstitial suppurative nephritis with an associated anomaly of the urinary tract, still one has to make a further differential diagnosis from a tuberculous kidney, renal calculus, and a kidney infection secondary to neuromuscular dysfunction of the bladder and ureter. This differentiation may be made clinically and verified by pyelography and flat x-ray pictures. Clinically it is rather difficult to differentiate between a tuberculous kidney and one of the obstructive type. In the five cases of tuberculosis of the kidney admitted to the Hospital for Sick Children during the course of this study, there were symptoms, such as intermittent pain, pyuria, and frequency, all pointing to involvement of the urinary tract. Only two gave a history of having short febrile periods. In these five cases there were definite tuberculous processes elsewhere, such as in the knee, spine, and lung, which would make one suspect the presence of a tuberculous focus in the kidney. Mantoux tests were strongly positive and the guinea pigs which were inoculated with these urines died of tuberculosis. The pyelograms in these cases were typical of tuberculous kidney, showing a dilatation or a moth-eaten appearance of pelvis. Renal calculus is seen fairly often in childhood, there being twelve cases admitted to the Hospital for Sick Children in the period quoted. Seven of these patients were over two years of age, and in these abdominal pain was the most common complaint. Hematuria was found in only two instances while pus was usually found in the urine at irregular intervals; in two cases it was persistent. The diagnosis of renal calculus was confirmed by flat x-ray pictures or pyelography. In five of these cases there has been associated hydronephrosis. There have been only two cases of neuromuscular dysfunction, and though clinically it cannot be differentiated from the obstructive type, still it is readily recognized on x-ray examination. In this type of case a cystogram is of undoubted value in showing up the greatly dilated bladder and reflux up the megaloureter to the pelvis of the kidney.

METHOD OF INVESTIGATION

All patients with pyuria admitted into the hospital are examined by means of intravenous urography. At first uroselectan was used, but now the more concentrated preparations, uroselectan B and neoskiodan, are used. The results of the x-ray plates seem to be about the same with either of the two last named preparations, but with the neoskiodan,

flushing, mouth dryness, and pain at the site of injection occur less frequently. In children over two years of age there is visualization of the urinary tract in about 95 per cent of the cases though the dosage of the drug used in this investigation was much higher than that prescribed by the manufacturer. It has been found that a child of two years of age and over will tolerate from 15 to 20 c.c., or practically the amount recommended for injection in the adult. So far there have been no harmful reactions to these large doses. When using the recommended dosage of 2 to 3 c.c. in children under two years of age, x-ray visualization has been very poor because of poor concentration of the drug in the urinary tract. At present better x-ray visualization has been obtained with larger doses, and as much as 1 c.c. per pound of body weight up to 10 pounds has been used. However, in these younger children and infants intestinal gas quite frequently obscures the urinary shadow, and at times this is rather difficult to dispel. When an anomaly is found, a cystoscopic examination with retrograde pyelograms is made.

In Table I are seen the x-ray findings localizing the site of obstruction.

TABLE I

SITUATION OF OBSTRUCTION

Place	*Right*	*Left*	*Bilateral*
a. In Ureter			
At Pelvoureteral junction	6	4	
1 to 1½ inches below pelvis	2	1	1
Pelvic brim	2		1
Below pelvic brim		1	
Ureterovesical orifice	3	8	4
Other places in ureter	3	2	1
b. In bladder			0
c. In urethra			1

From Table I it will be seen that in every one of the forty cases of pyuria, an obstruction was found. The site most frequently affected was the ureterovesical orifice.

TYPES OF OBSTRUCTION

Partial stenosis varying from a mild to a severe degree is the most commonly found type of obstruction. The production of this stenosis has been explained by various observers. Englisch[2] in 1879 reported the anatomical structure of the urinary tract in an unpicked series of 1,200 infant cadavers. He found three normal sites of narrowing of the ureter—at the pelvoureteral junction, at the iliac crest, and at the juxtavesical areas of the ureter. In sixty-five cases he found an exaggeration of this normal physiologic narrowing. Of these, thirty-four were at the pelvoureteral junction; twenty-eight were at the juxtavesical narrowing, while only three were found at a site between these two places. Findings of a similar nature were published by Robinson[3] in

1904. He examined 200 ureters filled with air, with paraffin, and with radioopaque substances. He also found three zones of widening and three of narrowing, the latter being in the pelvoureteral junction, the iliac and the juxtavesical regions of normal ureters. Schrieber[4] in his study of 100 consecutive autopsies has found stricture of the ureter existing as a definite pathologic entity in 12 per cent of the cases examined. He believes that the etiologic factor of stricture or stenosis is due (a) to a congenitally accentuated narrowing of a congenital physiologic narrow site; (b) to extension of inflammatory processes into the ureteral wall from adnexal disease, with and without thrombophlebitis and advanced cystitis; and (c) to kinking and compression due to crossing of anatomical structures, namely, vas deferens in the male and uterine artery in the female. Schrieber does not believe the stricture is due to a localized intrinsic inflammatory process in the ureteral wall which might have resulted from a focus of infection.

In this series the types of obstruction found are given in Table II.

TABLE II

TYPES OF OBSTRUCTION FOUND AT DIFFERENT SITES

LOCATION	NO. CASES	CAUSE
1. At pelvoureteral junction	5	Partial stenosis
	5	Aberrant vessels
2. At 1 to 1½ inches below pelvic brim	4	Fibrous bands
3. At pelvic brim	3	Partial stenosis
4. Below pelvic brim	1	Fibrous band
5. At ureterovesical orifice	15	Partial stenosis
6. Other places in ureter	6	Double ureter
7. In urethra	1	Membranous urethra

In Table II it is seen that in twenty-three cases there was an obstruction due to partial stenosis. In the five cases there were fibrous bands which at operation seemed to be of a congenital formation rather than of an inflammatory nature. Of the six cases of double ureter there occurred a dilatation of the ureter to the lower pole and a hydronephrosis of the lower pelvis of the kidney; the obstruction appeared to be at the junction of the two ureters in these cases. In two cases the splitting of the ureter was complete. In one case of complete splitting of the ureter, there was found at operation an obstruction of the ureter to the lower pole of the kidney, caused by the crossing of the ureter from the upper pole one-half an inch above the bladder. Ptosis of the kidney and kinking of the ureter have been found but are probably secondary to the dilatation that has been formed in either the pelvis or the ureter.

In three cases more than one type of anomaly was present:

a. Double ureter right side and aberrant vessel left side.

b. Left aberrant vessel and right renal calculus.

c. Left renal calculus and right aberrant vessel.

In this series there have been three cases with associated renal calculi.

It will be seen in this series that the cause of obstruction has differed somewhat from those that Schrieber found. In twenty-three cases there was obstruction due to stenosis of the ureter, but the remainder had an extrinsic cause of obstruction, such as aberrant vessel, fibrous bands, split ureter, and membranous urethra.

TREATMENT

The treatment of chronic pyuria varied and has depended upon the type, cause, severity, and site of the obstruction. In cases of stenosis, especially at the ureterovesical orifice and lower one-third of the ureter, dilatation was accomplished by ureteral catheter. In the pelvoureteral junction the stenosis is dilated by surgical intervention. The kidney is exposed, pelvis nicked, and a dilating catheter passed through this and down the ureter past the obstruction. Aberrant vessels were ligated and cut. Fibrous bands were removed and in cases of severe ptosis a nephropexy is performed. In a few cases where there has been severe kidney infection or destruction, nephrectomy was done. In Table III is found the treatment and the immediate results.

TABLE III

IMMEDIATE RESULTS OF TREATMENT

TREATMENT	NUMBER OF CASES	NO FURTHER URINARY SYMPTOMS	IMPROVED
None after original ureteral catheterization at the cystoscopic investigation	12	6	6
Dilatation of obstruction	12	4	8
Laparotomy with treatment found necessary	10	2	8
Nephrectomy	5	5	
Section of ureterovesical orifice			1

In Table III the immediate results are based on the condition of the child on discharge from the hospital, being from two to four months after operation, and the points considered included recurrence of any urinary symptoms and the degree or absence of pyuria at that time. In seventeen cases the child was discharged with negative urinalysis, while the remainder still had some indication of kidney infection but were improved in comparison with the condition present on admission.

As to the ultimate outcome of these cases, it is rather difficult to draw any definite conclusions at the present time. Sixteen of these cases have been followed in the Out-Patient Department over a period ranging from four months to three years, and the results found are tabulated in Table IV.

If the results are to be judged on the disappearance of pus from the urine as the criterion for the cure of the kidney infection, then it can be said that there is a definite improvement, but not a cure. But there are other things to be taken into consideration, such as recurrence of

TABLE IV

TREATMENT	RESULT
No treatment except catheterization on cystoscopic examination	(1) 6 mo. later occasionally a urinalys's showed few white blood cells per H.P.F. No febrile reaction. (2) 1½ yr. later, albumin positive, 4 to 6 pus cells per H.P.F. No febrile attack.
Dilatation	(3) 4 mo. later urine negative. 1 yr. later 0 to 1 white blood cell. No febrile reaction. (4) 5 mo. later persistence of pus after immediate improvement. Nephrectomy done. 2½ yr. later no symptoms, no pus. (5) 1 yr. later occasional white blood cell. No fever. (6) 1¼ yr. later having repeated dilatation, mostly pus-free urinalysis. (7) 1½ yr. later no pus till this time, then acute attack of pyuria; pus + + ; albumin, trace; redilated and only one pus cell found during next year. (8) 2½ yr.—mostly negative urinalysis, then mild attacks. Redilated.
Operation	
(9) Aberrant artery	1 yr. later pain but no pyuria; nephropexy. 2 yr. later no pyuria; no hydronephrosis.
(10) Aberrant artery	1 yr. later no symptom.
(11) Separation of double ureter near bladder	6 mo. later negative urinalysis. 1 yr. later 4 to 8 white blood cells. No symptoms.
(12) Separation of double ureter—upper ureter obstructing lower ureter	No symptoms for 2 yr. then acute attack. 3 yr. later occasional white blood cell and trace of albumin.
(13) Obstruction 1½ in. below pelvis dilated from above and nephropexy	6 mo. later no pyuria, no symptoms.
(14) As above (13)	8 mo. later, mostly negative urinalysis, but occasional white blood cell.
(15) Removal of fibrous bands	2 wk. later, occasional white blood cell. 6 mo. later, no hydronephrosis, occasional white blood cell. 1 yr. later, trace of albumin, occasional white blood cell, no fever in this time and general health good.
(16) Proximal 1½ in. ureter in S-shaped kink and adherent to kidney pelvis—relieved	1 yr. later mild pyuria still present, but no fever or symptoms. Albumin beginning to appear.

acute attacks with fever, the disappearance or prevention of further hydronephrosis, and the general health of the child. In only three cases was there recurrence of fever with increased pus in the urine, and in one other case, though fever was absent, the pyuria was marked. In the remaining thirteen cases the general health was better as indicated by gain in weight, good appetite, lack of tiredness, and disappearance of symptoms referable to the urinary tract. In the patients recytoscoped there was either disappearance of the hydronephrosis or delay in further destruction of kidney tissue, as indicated by the hydronephritic sacs remaining the same size.

Medical treatment, such as prolonged alkalinization with potassium citrate and sodium bicarbonate, and treatment by means of ammonium

chloride have been tried with no improvement. For the past five months there have been nine cases of persistent pyuria in patients on ketogenic diets.[5] In these the immediate results have been good. The urine has become both pus-free and bacteria-free. Even after they were followed several months in the Out-Patient Department, there have been no recurrences of symptoms, and the urine has remained bacteriologic and pus negative. These children have gained weight. At present, however, we do not know what the end-results are going to be or if destruction of kidney tissue is still continuing although signs of infection have disappeared.

UNTREATED CASES

During the past three years there have been admitted into the hospital three cases of long-standing persistent pyuria. In each case there has been a similar history of persistent pyuria of over twelve, seven and six years' duration, respectively. There was found in the admission examination a high blood pressure, arteriosclerotic vessels, and eye-ground changes consisting of albuminuric retinitis. Albumin from 2+ to 3+ was the outstanding characteristic of the urinalyses, though a few white blood cells and a few casts were found. One patient had intermittent hematuria which lasted only a few days at a time. There was no edema; headaches were common. Determinations made on the blood for N.P.N. and creatinine gave high values. In these three cases an obstruction was found. In one case there was obstruction in both ureters, but though repeated dilatations were performed, the case progressed and ended fatally. Apparently secondary changes had occurred in the kidney before treatment was instituted. The same thing happened in a second case, but in this there was bilateral stenosis of the ureterovesical orifices. Section of the orifices was done, but though the orifices were still widely dilated three years later, the child died of a secondary chronic interstitial nephritis. In this case there had been no further increase in the size of the ureters and pelves of the kidney. The third case was one in which the obstruction was due to a membranous urethra, but no operative treatment was attempted as the child had a severe secondary interstitial nephritis. Thompson,[6] in 1927, has reported a similar series of chronic nephritis with obstruction in the lower urinary tract.

The above three cases show the end-result of chronic interstitial suppurative nephritis which may occur in an obstruction of the urinary tract. To prevent such a condition we feel that it is essential that some form of treatment be attempted. At present, relief of the obstruction seems to be the logical method although time may prove that the ketogenic diet will be efficacious. With relief of the obstruction there is allowed greater drainage from the infected kidney. Besides this, any

further dilatation of the pelvis of the kidney is prevented; this in turn prevents further pressure atrophy of the kidney substance and lowering of the natural resistance.

SUMMARY

1. The results of the study of forty cases of persistent pyuria are presented. This investigation has included clinical, intravenous pyelography, and cystoscopic examination.

2. From a clinical point of view a complete urologic investigation is indicated in any case of pyelitis in which the urine shows pus over a period of four weeks' time. The investigation has shown that the great majority of cases of chronic pyuria are due to inadequate drainage due to an obstruction of the urinary tract. The treatment has been directed to relief of the obstruction present. The immediate results are fairly satisfactory, but the end-results as far as kidney infection is concerned are indefinite. In these treated cases, though there may be recurrence of pus in the urine, there is noted a definite improvement in the general health of the patients.

REFERENCES

1. Summerfeldt, Johnston, and Kaake: J. Pediat. 2: 493, 1933.
2. Englisch: Deutsche Ztschr. f. Chir. 2: 11, 1879.
3. Robinson: Am. J. Derm. and Genito-Urin. Dis. 8: 106, 1904.
4. Schrieber: Surg. Gynec. Obst. 45: 423, 1927.
5. Helmholz, H. F.: J. A. M. A. 99: 1305, 1932.
6. Thompson: Guy's Hosp. Rep. 77: 461, 1927.

CHRONIC PYURIA (CONGENITAL DEFECT)—PRESACRAL SYMPATHECTOMY

Case Report

Pearl Summerfeldt, M.B., and Alan Brown, M.D.
Toronto, Ontario

THIS is a case of persistent pyuria due to a neuromuscular dysfunction of the bladder for which a presacral sympathectomy was done. We thought it would be of interest to report the findings, as we know of only one other on record.

A female child, thirteen and one-half months of age, was admitted to the Hospital for Sick Children Jan. 3, 1934, with a history of puffiness around the eyes shortly after birth. Outside of this, the child progressed favorably until she was six months old when she developed an acute illness with high fever, vomiting, and loose, watery stools. On the third day of her illness she began to cry on voiding, and on the fifth day she had two convulsions. A diagnosis of acute pyelitis was made on finding pus in urinalysis. During the next three weeks the child was very ill, received a blood transfusion, and was put on alternating alkali and acid therapy. From then the child slowly improved, but in the three months previous to her admission here she had had three recurrences of high fever and pus in the urine. The mother did not know if pus was present in between attacks or not. During the last attack the child was seen by one of us, who advised a complete urologic examination. There was nothing of importance in family history or in feeding history.

On physical examination the child weighed 18 pounds 4 ounces, appeared pale, moderately ill. The throat was slightly congested. Examination of the head, chest, and abdomen was negative, and the kidneys were not palpable. The vulva was scalded. On admission the urinalysis was alkaline in reaction, albumin +, and there were from 20 to 40 W.B.C. per high power field with some clumps of pus cells. The white blood count was 12,300; polymorphonuclears, 54 per cent; N.P.N., 35 mg.; and creatinine, 1.25 mg. Culture of the urine grew a *B. coli communior*.

A cystogram was made the day after admission. This consisted of catheterizing the bladder and injecting by gravity a 12.5 per cent solution of sodium iodide into the bladder. In this case about 60 c.c. was injected without any difficulty, and a flat x-ray plate was made (Fig. 1). In this there was found a reflux up the left ureter indicating a relaxed ureterovesical orifice, a left hydroureter, and hydronephrosis. There was no reflux up the right side. Following this, 15 c.c. of neoskiodan were given intravenously. The plates were rather indistinct, but the right pelvis appeared smaller than the left. A diagnosis was made of neuromuscular dysfunction of the bladder and left ureter. An attempt was made to treat the child by the use of the ketogenic diet. The diet at the beginning had a ratio of Fatty Acid : Glucose :: 2.1 : 1, and this was gradually worked up to Fatty Acid : Glucose :: 2.8 : 1. On this the pH of the urine dropped to 5.4. On urinalysis there was a trace of albumin from 3 to 12 W.B.C., and acetone, 4 plus,

Read at the Twelfth Annual Meeting of the Canadian Society for the Study of Diseases of Children, Brockville, June, 1934.

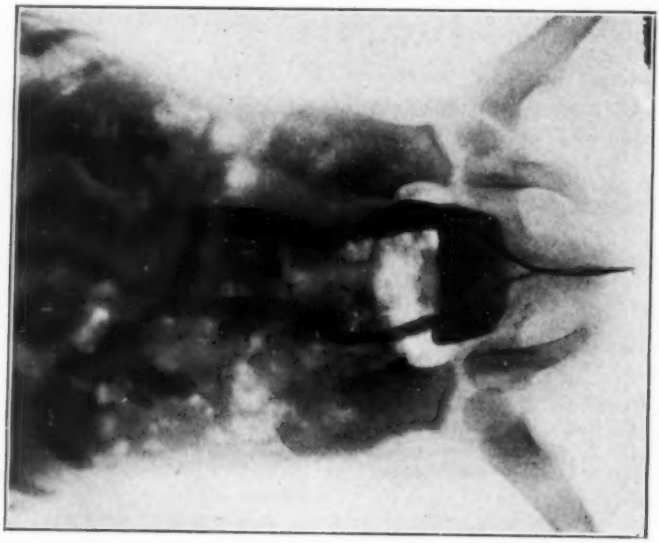


Fig. 2.—Pyelogram, Jan. 16, 1934.

Fig. 1.—Cystogram, Jan. 4, 1934.

but there was very little effect on the number of organisms present. There was some loss of weight, and the child vomited occasionally. Two weeks after admission the child had a cystoscopic examination (Fig. 2), which verified the findings on the left side and also showed moderately dilated right ureter and pelvis, but functioning ureterovesical orifice. At this time there was an acute recurrence of the pyuria which lasted ten days. During this period it was decided to discontinue the ketogenic diet and do a presacral sympathectomy for the left side. This was done on February 3 by Dr. Wansbrough of the surgical service.

Following the operation the child ran an irregular fever from 99° to 102° F. for four weeks; she developed furunculosis; and on urinalysis there were 25 to

Fig. 3.—Cystogram, Feb. 21, 1934. Taken seventeen days following operation.

100 W.B.C. per high power field and acetone, 4 plus. Ketogenic diet was resumed two weeks after the operation for a four-week period, and during the last week of this period the urine contained from 0 to 10 W.B.C. per high power field, acetone, 4 plus, and innumerable organisms. At this time she developed an upper respiratory tract infection with otitis media and recurrence of pus in the urine. Two weeks later when the child was ready for discharge, an acute intestinal obstruction occurred for which laparotomy was done. The patient was discharged three weeks later. At this time there was a faint trace of albumin, 1 or 2 W.B.C. per high power field, and organisms were still present in the culture.

Following the operation, cystograms and intravenous pyelograms were made at various intervals. Fig. 3 is the x-ray picture taken two weeks after operation. The bladder capacity was about 50 c.c. when an overflow took place. Left side un-

changed from original and there was slight reflux up right side. Two months after the operation an intravenous pyelogram showed that both kidneys were excreting well but the left side was larger than right. Fig. 4 is the x-ray picture of the cystogram taken a few days following the intravenous pyelogram. Bladder capacity was 40 c.c. Reflux was still present, but upper third of ureter was definitely smaller. Reflux of right side was noted, but the size of ureter and pelvis was just the same as in x-ray plates taken at the first cystoscopic. The child was seen in the outpatient department three weeks after discharge. The urine still contained a few pus

Fig. 4.—Cystogram, Apr. 2, 1934. Taken a few days after intravenous pyelogram.

cells, but no albumin was present. There had been no recurrence of fever and the child was gaining well.

In the case presented we have dilated ureters with no apparent obstruction to cause the dilatation, and on the left side the ureterovesical sphincter was relaxed. Following the presacral sympathectomy, there was decrease in size of bladder and ureter. At present we are only aware of one other case that has been published, with similar treatment. This was published by Ward[1] in the St. Bar-

tholomew Hospital Report. His patient had bilateral hydronephrosis, hydroureter with no obstruction. There was bilateral kidney infection. One month after operation the ureters were about one-half as wide as before operation except the lower ends, which were still markedly dilated. A second uroselectan was given eleven months after operation, and the right side appeared practically normal although there was still seen dilatation of lower end of left ureter. At this time the urine still showed some pus and numerous *B. coli*.

In recent years a great deal of interest has been attached to the sympathetic and parasympathetic nerves, especially those that innervate the lower half of the colon and pelvic viscera. Excision of the sympathetic, especially the presacral or hypogastric nerve, has been done for a number of conditions, as constipation, megacolon, atony of the bladder, dysmenorrhea, and pelvic pain.

The sympathetic system is derived from the second thoracic to the third lumbar, inclusive. From the lumbar sympathetic chains, there arise on each side four nerves which pass outward to join each other and thus form a long nerve pathway from the first lumbar ganglion to the hypogastric ganglion. The hypogastric ganglion or presacral nerve is an irregular fenestrated nerve. It sends branches to the ureter, internal sphincter, superior part of the rectum, bladder, seminal vesical, and ductus deferens. The hypogastric ganglion is not purely a sympathetic nerve, as there are parasympathetic fibers from the third and fourth sacral nerves.

The function of the hypogastric plexus or presacral nerve is not fully understood. Learmouth[2] found that the action of the hypogastric plexus was to produce closure of the internal sphincter, to cause the ureters to efflux their contents into the bladder, and to oppose the contraction of the detrusor muscles of the bladder, which are activated by the parasympathetic. It is therefore sometimes spoken of as the filling nerve of the bladder in contrast to the emptying powers of the parasympathetic. Thus, stimulation of the sympathetic will cause contraction of the sphincters of the bladder and rectum with dilatation of the viscera, while stimulation of the parasympathetic causes the opposite effect—dilatation of the sphincter and contraction of the visceral wall to allow emptying. This has been proved experimentally in the cat by Adamson and Aird.[3] The parasympathetic to the hypogastric ganglion was cut, which resulted in immediate retention of urine. Following this, there was marked progressive dilatation of both bladder and colon.

Clinically there has also been found an association between the bladder and megacolon. Adamson and Aird[3] have found in 5 per cent of their megacolon cases a dilatation of the bladder. Also, N. M. Dott found in four cases of megacolon three with a residual urine and

dilated bladder. After presacral sympathectomy for the megalocolon, the bladder condition showed improvement.

In the case presented we have primarily a congenital neuromuscular dysfunction or unbalance between the parasympathetic and sympathetic —the sympathetic being overactive. This has resulted in a moderate dilatation of the bladder, left hydroureter, with a moderately severe left hydronephrosis, which has resulted in a pressure atrophy of the kidney substance. With this condition retention of urine has occurred in bladder and left urinary tract, producing a passive congestion in the kidney, which has lowered the resistance of the kidney to any organism that might be present. Medical treatment over a long period of time had been of no avail and the ketogenic diet in the hospital had not killed the organism.

At the present time we are unprepared to make any definite conclusion as to the outcome of this treatment. So far there has been produced an increase of tone in the bladder and, to a slight extent, in the ureter; this may relieve the stasis that was present. As far as the kidney infection is concerned there has been no improvement whatever.

SUMMARY

A case of persistent pyuria in a female child, thirteen months old, is presented. This had lasted for several months and was due to a neuromuscular dysfunction of the left ureter and bladder. The case was treated by doing a presacral sympathectomy. One month following the operation, there was a diminished capacity of the bladder and a decrease in the size of the upper third of the left ureter. On discharge the urine still contained organisms, a faint trace of albumin and 1 or 2 W.B.C. per high power field.

REFERENCES

1. Ward, Ogur R.: St. Barth. Hosp. Rep. 66: 17, 1933.
2. Learmouth, James R.: J. Urol. 25: 531, 1931.
3. Adamson, W. A. D., and Aird, I.: Brit. J. Surg. 20: 220, 1932-33.

AMEBIASIS IN CHILDREN

SAMUEL J. NICHAMIN, M.D., HENRY G. PONCHER, M.D., AND
MARION HOOD
CHICAGO, ILL.

AMEBIC dysentery with its classical gastrointestinal symptomatology is relatively uncommon in infancy and childhood. DeBuys[1] reported that out of 313 cases of amebic dysentery admitted to Charity Hospital, New Orleans, only four occurred in children under twelve years of age. Dougherty[2] stated that in the Henrietta Eggleston Hospital for Children in Atlanta only one case of amebic dysentery occurred in 3,180 admissions, including over 200 cases of diarrhea.

The prevalence of infection with *Endameba histolytica* cannot, however, be gauged by the incidence of one clinical form of amebiasis. Amebic dysentery is only one of the more severe clinical expressions of the infection while the term amebiasis represents the state of infection in man by *Endameba histolytica* regardless of the nature or degree of clinical manifestations. This differentiation is an important one. If the incidence of amebiasis in infants and children were judged by the number of cases of amebic dysentery one would obtain a false impression of the clinical and public health problem. That such problems actually exist was evidenced in a careful study of patients admitted to our dispensary this past year. The recent Chicago epidemic has served to make the medical profession, especially in the northern parts of the United States, "amebic conscious." As a result there has been a relearning and a realignment of preconceived impressions about the disease. The unsuspected finding of *Endameba histolytica* in the stools in a few cases that presented vague abdominal symptoms simulating chronic appendicitis prompted us to investigate further other cases in children presenting similar complaints. No attempt was made in this study to conduct a systematic study or survey of the disease.

It has been definitely established by numerous investigators that amebiasis is prevalent throughout the world and should not be considered as solely a tropical disease. In the United States most of the reports on the incidence of the infection have come from the southern states and Pacific Coast. In an analysis of cases of amebiasis in the

From the Departments of Pediatrics and Internal Medicine, College of Medicine, University of Illinois, Chicago.

hospitals of thirteen southern cities Dougherty[2] found that clinical amebiasis occurred in children under fifteen years of age in 8 per cent of the cases. The incidence of infection with *Endameba histolytica* in children from one month to five years of age was 16.5 per cent, and from six years to fifteen years, 20 per cent, according to Faust[3] in a survey undertaken in southwestern Virginia. The percentage of infection varied greatly in different localities. Milam and Meleney[4] found an incidence of 15.4 per cent of infection under one year of age, 27.5 per cent from one to four years; and 30.5 per cent from five to nine years in an epidemiologic study of amebiasis in a rural community in Tennessee. Sumerlin[5] found an incidence of infection with *Endameba histolytica* of 0.4 per cent among 513 children in private practice in San Diego, California. It is thus seen that the incidence of amebic infection in children varies widely with the economic status of the patients and the locality.

Statistics covering the incidence of infection with *Endameba histolytica* in children for the northern parts of the country have been very meager. Owen, Honess, and Simon[6] recently reported a survey for intestinal protozoa in eighty-three American Indian boys living in a mission school on a reservation in central Wyoming. They found *Endameba histolytica* in 26.5 per cent of the total number of cases examined. Owen[7] also investigated children in an orphanage in Oregon for protozoan infestations. He found in a group of forty-two boys a protozoan infestation of 71 per cent, 4.7 per cent of which were positive for *Endameba histolytica*. Tansinsin[8] found a percentage of infection with *Endameba histolytica* of 3.0 per cent in 115 children in Pennsylvania. This percentage was based on only one laboratory examination on the stool specimen of each patient for *Endameba histolytica*. Boeck and Stiles[9] found a percentage of infection with *Endameba histolytica* of 17.6 in the age group of five to nineteen years in training schools in Washington, D. C.

Obviously such data concerning restricted groups living under supervision with well-established sanitary and hygienic practices are of limited value. No accurate conclusions can be drawn from them as regards the general incidence of infection in children with *Endamebic histolytica*. However, they do indicate that there must be an appreciable percentage of infection in children in these areas. In adults residing in the northern states the percentage of infection with this parasite has been found to vary between 4.5 per cent and 22.5 per cent according to Sistrunk,[10] Giffin,[11] and Sanford,[12] each of whom reported surveys from the same institution in different years. Recognized authorities have estimated that from 5 to 10 per cent of individuals in this country are infected with *Endameba histolytica*.

There is undoubted and established evidence as to the pathogenicity of this parasite for man. Invasion of the intestinal tissues probably

occurs in all individuals harboring the parasite, the extent of the lesion being dependent upon the various factors that influence the natural resistance of the host, such as improper food, overwork, repeated exposure to infections, climate, etc. Stuart[13] called attention to the theory that there is a biologic equilibrium established between host and parasite. Acute cases are rare and indicate a lack of equilibrium. These may develop acute symptoms after surgical operations, shock, malnutrition, and causes that undermine the general well-being of the patient. In the tropics where the incidence of amebas is high, acute cases are usually associated with bacillary dysentery. In the epidemiologic report of the League of Nations[14] it was felt that the problem of acute cases of amebiasis seemed to depend upon the determination of the efficient causes of dysentery among the ameba carriers, both in temperate and warm regions, or the causes for the change of the amebas from a saprophytic to a parasitic mode of existence. The opinion was expressed that such a change was most probably brought about by a cessation of tolerance on the part of the human host.

Because of the natural resistance of a great majority of the individuals harboring *Endameba histolytica*, the minute lesions continually being produced are rapidly healed so that the clinically recognizable symptom-complex associated with amebic dysentery does not develop. Such individuals have been called "carriers" with the implication that the parasites are nonpathogenic and produce no lesions in their intestinal tract. The weight of clinical, pathologic, and experimental evidence is certainly opposed to such a view. In fact, Craig[15] has repeatedly stated that the great proportion of so-called "carriers" of *Endameba histolytica* present symptoms some time during the course of their infection. Strictly speaking it is preferable to consider the carrier state in amebiasis as latent amebic infection in which the symptoms of the presence of the parasites may or may not impress themselves on the consciousness of the host.

There seems to be no definite correlation between the extent of the intestinal lesions, which may range from minute defects microscopic in size to extensive ulcerations, and the severity of the clinical symptoms. Marked amebic ulcerations, and even amebic liver abscesses, have been repeatedly found in individuals who presented no classical symptoms of infection with *Endameba histolytica*. It follows, therefore, that the presence of *Endameba histolytica* in the intestinal tract of an individual is a far from harmless condition and is a potential danger to the well-being of the host.

Disturbances of health of varying degrees have been reported by many investigators in most of the patients with amebiasis. On the whole, their complaints have been associated with disturbances in the

gastrointestinal tract; chiefly constipation with occasional attacks of mild diarrhea, vague, colicky pain, especially in the right lower quadrant, with tenderness present both diffusely or over the colon, anorexia, and loss of weight. Headaches, irritability, lethargy, and neuralgic pains in the extremities have also been described in latent amebic infections.

In the past few years the above mentioned considerations regarding the incidence of infection and the pathogenicity of *Endameba histolytica* have been often stressed by students of this disease; they have pointed out to the medical profession that an amebiasis problem does exist in this country. The importance of recognition and treatment of the so-called "carriers," who are responsible for spreading the *Endameba histolytica* in the form of cysts (the only infective forms of the ameba), is obvious from the standpoint of both the patient and the general public. Just how this problem should be attacked has not been definitely decided by local and state health authorities. Obviously, the epidemiologic aspects of amebiasis cannot be adequately dealt with until the incidence of the infection is known and some of the disputed points regarding the pathology of the carrier state are definitely settled.

Clinically, the attention of the profession has been focussed for the most part upon the adult population. The importance of the problem of latent amebic infection in infancy and childhood has not been adequately appreciated. This has been partially due to unfamiliarity of many practitioners with certain phases of amebic infection or amebiasis. As aforementioned, a few surveys have been made to determine the incidence of infection of children in various communities in the United States. In addition there are a number of reports in the literature recording cases of classic amebic dysentery in infants and children. No attempt, however, has thus far been made to elucidate and emphasize the milder symptoms with relation to the gastrointestinal tract that may occur in children as a result of infection with this parasite, aside from the familiar dysenteric symptoms. Undoubtedly children are as susceptible to the infection as adults, and it is questionable whether they are less exposed to the etiologic factors in this disease. The realization of the prevalence of amebic infection in this country, together with the knowledge of its pathogenicity, should lead more often to a consideration of amebiasis in the differential diagnosis relating to obscure or vague abdominal symptoms in childhood.

Owen, Honess, and Simon[6] stated that their data in Indian children which revealed the high incidence of infection with *Endameba histolytica* of 26.5 per cent should prompt the physician to "bear in mind the possibility of the presence of *Endameba histolytica* in making a diagnosis of all obscure intestinal complaints." Kessel and Mason[16] made a study with a view toward comparing laboratory and clinical

observations in protozoan infection of the human gastrointestinal tract. Among their conclusions they noted that patients harboring *Endameba histolytica* had colitis symptoms about three times as frequently as those in whom the parasites were not found. They therefore stressed the importance of routine stool examinations for *Endameba histolytica* in patients exhibiting intestinal symptoms.

The causes of obscure abdominal pain in children are many and of diverse origins. A large percentage of cases presenting this symptom are due to simple and benign causes such as enterospasm and meteorism as a result of dietary indiscretion.[17] There is reason to believe, however, that intestinal parasites have seldom received adequate consideration except in regions where infestation is endemic. In many cases where amebiasis has been considered, the care and method of collection of the stool specimen would certainly make for a negative report. It is only after many careful examinations by experienced workers on a freshly passed, warm stool that the diagnosis of amebiasis can be ruled out. Craig[18] states that at least six specimens of feces collected at different times should be examined before a negative result can be regarded as final. It has also been emphasized by many workers that a single stool examination reveals only about one-third of the total incidence of intestinal protozoa. This percentage might be increased if, as Sumerlin[5] points out, liquid stool specimens following a saline cathartic are examined while warm. In the majority of cases in children the diagnosis of amebiasis cannot be made without the finding of parasites in the feces. The clinical symptoms of vague abdominal pain associated with constipation or diarrhea are far from characteristic or pathognomonic of any one intestinal or abdominal ailment. Obscure types of abdominal pain in children occurring as a part of the variable symptomatology of amebiasis may simulate many abdominal conditions. Appendicitis is one of the most frequent diseases confused with the vague gastrointestinal symptoms of amebiasis. This is a common problem for differential diagnosis in the tropics, or wherever amebiasis is found. In two of the cases of our series appendectomies had been performed and later, after careful search, *Endameba histolytica* were found. In a third patient the parasites were discovered while operation was being contemplated. In the following cases the diagnosis of amebiasis was established only after diligent search for the parasite was made. In several cases, two or three examinations were necessary, and in one case the parasites were noted for the first time on the fourth examination.

It is of interest to note that, in addition to *Endameba histolytica, Endameba coli* cysts were found in all of our patients. The latter were especially noted in those stool examinations preceding the specimen which finally revealed the pathogenic parasite. Such an association between a pathogenic and nonpathogenic parasite is more than of aca-

demic interest. Various representative surveys in different parts of the world in which the comparative incidence of *Endameba coli*, *Endameba histolytica*, and *Endolimax nana* were noted were analyzed by Faust.[19] Such a study revealed a definite correlation between the *Endameba histolytica* index and *Endameba coli* and *Endolimax nana* index. A low *Endameba histolytica* index was usually associated with a relatively low *Endameba coli* and *Endolimax nana* index. Faust[19] accumulated additional data from various regions and showed that in about 60 per cent of the cases in which *Endameba histolytica* was noted, *Endameba coli* was also found. He concluded that the *Endameba coli* index of a community was a relatively accurate indication of the amount of *Endameba histolytica* in that area. He further suggested the possibility of considering seriously the presence of *Endameba histolytica* whenever *Endameba coli* was found.

The following case reports are representative of some of the clinical problems of amebiasis in children.

CASE REPORTS

CASE 1.—A. M., white female patient, aged twelve years, was admitted to the hospital on Feb. 27, 1933, with the history of pain of one year's duration in the right lower quadrant, occasionally associated with fever and vomiting, occurring about every month and lasting about a week. She passed pinworms three or four times in the past two years. Bright red blood was noted in the stools on several occasions. The physical examination was essentially negative except for slight tenderness of the abdomen on deep pressure over the right lower quadrant, right upper quadrant, and epigastrium. Rectal examination was negative. White blood count was 10,550; polymorphonuclears, 41 per cent; monocytes, 7 per cent; lymphocytes, 52 per cent; and eosinophils, 2 per cent. Patient was discharged and referred to the out-patient department for further laboratory and clinical examination. The findings remained essentially negative, and it was felt at that time that a diagnosis of chronic appendicitis was very questionable and that surgery was not indicated. About five months later, Aug. 30, 1933, she was readmitted to the hospital on the surgical service with the complaint of pain in the right lower quadrant of twenty hours' duration, nausea, but no vomiting. Mother stated that the child had had several similar attacks during the past year. She presented acute tenderness in the right lower quadrant in the region of McBurney's Point, with no rigidity. The white blood count was 12,350. Appendectomy was considered advisable. The appendix was found long and kinked in the middle, but it did not appear to be acutely inflamed. Patient was discharged Sept. 9, 1933, in good condition. She was seen in the dispensary again one month later, at which time she complained of pain in the right lumbar region. Urine showed one plus albumin, occasional red blood cells, and numerous pus and epithelial cells. Blood examination showed a white blood count of 8,350; polymorphonuclears, 24 per cent; lymphocytes, 64 per cent; monocytes, 2 per cent; and eosinophils, 10 per cent. Stool examination was negative for ova and parasites. On later visits, tenderness on palpation over abdomen persisted. Repeated stool examinations revealed first *Endameba coli* cysts, and later *Endameba histolytica*. Patient received the routine treatment for amebiasis at the Municipal Contagious Hospital with complete subsidence of symptoms.

CASE 2.—B. J. H., white female, aged seven years, was admitted to the dispensary July 3, 1933, with complaint of abdominal pain of two years' duration, located

around the umbilicus. The pain was intermittent and cramplike in character; attacks occurred every few weeks, and were associated with nausea, occasional vomiting, and slight fever. Appetite was only fair, and bowel movements were regular. No attacks of diarrhea or bloody stools had been present. Recurring attacks of vague abdominal pain were noted at frequent intervals during the visits to the dispensary. Physical findings were essentially negative, except for tenderness on deep palpation over the right lower quadrant. In October, three months after the first visit to the clinic, the child still complained of pain in the right lower quadrant of abdomen with no new objective manifestations. At this time the gastrointestinal tract was examined with an opaque meal and revealed the appendix apparently adherent and kinked, with tenderness to pressure over it, and prolonged retention of its contents. The findings were consistent with pathologic appendix. Patient was then referred to surgery for an opinion as to the advisability of performing an appendectomy. Urine was negative. The white blood count was 15,150; polymorphonuclears, 29 per cent; lymphocytes, 63 per cent; monocytes, 5 per cent; and eosinophils, 3 per cent. Patient was seen in the surgical dispensary, and later admitted to the hospital on their service, Nov. 17, 1933, when it was noted that she had been having an exacerbation of the right lower quadrant pain over a two-week period. Appendectomy was performed on the following day. The appendix was found injected but not adherent. The patient made an uneventful recovery. In view of the fact that a sister of the patient had an attack of abdominal pain associated with the passage of bloody, mucoid stools in January of this year, the patient's stools were repeatedly examined for *Endameba histolytica*. Several examinations revealed only *Endameba coli* cysts. After administration of a saline cathartic on Apr. 24, 1934, *Endameba histolytica* was found. Patient was referred for treatment.

Case 3.—Mary G., white, aged twelve years, was admitted on the surgical service on Dec. 4, 1933, with complaint of pain in the right lower quadrant of abdomen for two days. In addition a history of vague, generalized abdominal pain of many months' duration was obtained. There was no relation to the ingestion of food or bowel movement. Vomiting occurred only occasionally. Constipation was noted, and patient had been given cathartics frequently. Physical examination was essentially negative, except for moderate tenderness on deep palpation over the course of the colon, more marked on the right side. There was no rigidity. Rectal examination was negative. The white blood count was 6,650; polymorphonuclears, 50 per cent; lymphocytes, 46 per cent; monocytes, 2 per cent; and eosinophils, 2 per cent. Fresh, warm stools revealed the presence of vegetative forms of *Endameba histolytica* with occasional *Endameba coli* cysts on repeated examinations. Proctoscopic examination revealed typical amebic ulcerations in the rectum. Patient was transferred to Municipal Contagious Hospital where she received treatment over a period of six weeks. Since then, child has been perfectly well and has had no complaints.

Case 4.—E. H., aged ten years (sister of B. J. H., Case 2), was first seen in the pediatric dispensary Jan. 15, 1934. Mother stated that the patient had been perfectly well until two days before admission when she woke up with severe abdominal pain followed by several vomiting spells. Three bloody, mucoid stools were passed during the day. The pain was intermittent and located around the umbilicus. Improvement occurred later in the day. Physical examination was negative except for slight exophthalmos and slightly enlarged thyroid. The white blood count was 10,800; polymorphonuclears, 60 per cent; lymphocytes, 28 per cent; monocytes, 12 per cent. Examination of stool at this time revealed no parasites. February 22, one month later, the vegetative forms of *Endameba histolytica* were demonstrated in addition to *Endameba coli* cysts. When this was written, patient was under treatment.

Another sister in this family, thirteen years old, had had her appendix removed four years ago for chronic appendicitis, according to the mother. Thus far only *Endameba coli* cysts have been found.

CASE 5.—M. A., white female, aged ten years, was first seen in the dispensary on Jan. 30, 1934, with the complaint of cramplike pains in the abdomen of three weeks' duration. One year ago she had an attack of diarrhea which lasted three weeks, the stools containing mucus but no blood. In addition she suffered from dizzy spells and nausea, which were associated with the generalized abdominal pain. There was no vomiting and no fever. Physical examination was essentially negative, except for tenderness on palpation and slightly increased resistance in the right lower quadrant of abdomen. Urine was negative. The white blood count was 7,650; polymorphonuclears, 57 per cent; lymphocytes, 37 per cent; monocytes, 3 per cent; and eosinophils, 3 per cent. During the following six weeks, the child continued to complain of vague pains in both lower quadrants of abdomen, especially on the right side. Stool examination showed encysted forms of *Endameba coli*. A second stool examination showed many giardia cysts, frequent *Endameba coli* cysts, and frequent Charcot-Leyden crystals. No *Endameba histolytica*, trophozoites, or cysts were found. One week later another stool examination showed similar findings. However, on the fourth examination a few days later a cathartic stool was obtained, and it revealed, in addition to the trophozoites, precystic forms, and cysts of *Endameba histolytica*. Patient was under treatment at the time this was written.

CASE 6.—Mae G., white, aged eight years, was admitted to the dispensary March 6, 1934. She was brought in for a general physical examination. However, on questioning, a history of vomiting soon after taking food, vague, generalized abdominal pains, and constipation was elicited. Physical findings were essentially those associated with malnutrition. The white blood cell count was 6,550; polymorphonuclears, 52 per cent; lymphocytes, 30 per cent; monocytes, 9 per cent; and eosinophils, 9 per cent. Stool examination showed numerous encysted forms of *Endameba coli*. Another stool examination, one week later, revealed frequent *Endameba coli* cysts and occasional *Endameba histolytica* cysts, in addition to Charcot-Leyden crystals. A cathartic stool specimen revealed the vegetative forms of *Endameba histolytica*.

SUMMARY

The available data on the incidence of amebiasis in children in the northern states and the recent evidence of amebic infection in and about Chicago warrant the attention of the medical profession to the problem of latent amebic infection in childhood. In the past few years the symptomatology and pathology in such infections in adults have been repeatedly stressed, especially by Craig. From the cases cited in this report it is apparent that children infected with *Endameba histolytica* may likewise manifest obscure and unexplained gastrointestinal symptoms which at times may lead to faulty diagnoses of acute or chronic appendicitis and other diseases of the alimentary tract. Careful and repeated examinations for parasites in the stools of children exhibiting vague abdominal complaints should be undertaken whenever possible.

REFERENECS

1. DeBuys, L. R.: J. A. M. A. 63: 1806, 1914.
2. Dougherty, Mark S., Jr.: Am. J. Trop. Med. 13: 317, 1933.
3. Faust, E. C.: Am. J. Trop. Med. 11: 231, 1931.

4. Milam, Daniel F., and Meleney, Henry E.: Am. J. Hyg. 14: 325, 1931.
5. Sumerlin, H. S.: J. A. M. A. 102: 363, 1934.
6. Owen, William B., Honess, Ralph F., and Simon, James R.: J. A. M. A. 102: 913, 1934.
7. Owen, W. B.: Northwest. Med. 31: 186, 1932.
8. Tansinsin, Manuel S.: Arch. Pediat. 47: 113, 1930.
9. Boeck, W. C., and Stiles, C. W.: Bull. 133, Hyg. Lab. U. S. Public Health Service, 1923.
10. Sistrunk, W. E.: J. A. M. A. 57: 1507, 1911.
11. Giffin, H. Z.: J. A. M. A. 61: 675, 1913.
12. Sanford, A. H.: J. A. M. A. 67: 1923, 1916.
13. Stuart, M. A.: U. S. Nav. M. Bull. 26: 411, 1928.
14. League of Nations. (General Review of Dysentery, Its Geographical Distribution, League of Nations Epidemiological Report.) March—April, 1933, pp. 48-86.
15. Craig, Charles F.: J. A. M. A. 98: 1615, 1932.
16. Kessel, J. F., and Mason, V. R.: J. A. M. A. 94: 1, 1930.
17. Schlutz, Frederick, W.: J. Pediat. 2: 41, 1933.
18. Craig, Charles F.: Internat. Clin. 1: 77, 1929.
19. Faust, Ernest Carroll: Am. J. Trop. Med. 10: 137, 1930.

1819 West Polk Street

AMEBIC ABSCESS OF THE LIVER

Report of a Case in a Child Five Years Old

Lewis K. Sweet, M.D.
Peiping, China

IN TROPICAL and subtropical countries in which amebic dysentery is endemic, amebic liver abscess is common in adults. Rogers[1] has given an excellent general discussion of the condition, and Ludlow,[2] Chen, Van Gorder, and Yuan,[3] Manson-Bahr[4] and others have added valuable observations on groups of patients. In children, however, the disease is rare. Legrand[5] in 1906 collected 112 cases of liver abscess from all causes in children. Of these thirty-nine were "dysenteric"; nineteen were traumatic; nine were of doubtful origin; and the remainder were pyemic. Other cases have been reported since that time, but in 1931 Manson-Bahr[4] wrote that the disease is "unknown or extremely rare in children." His youngest patient was sixteen years old. Likewise the youngest patients reported by Chen, Van Gorder, and Yuan,[3] Thurston,[6] and Murray and Koh[7] were seventeen, fourteen, and fourteen years of age, respectively. Ludlow, however, in 160 cases reported from Korea, included three children under the age of ten years, the youngest being two years old.

Among the youngest patients on record with the disease are those reported by Brown,[8] Niblock[9] and Le Roy des Barres.[10] Brown in 1824, in the earliest reference to the condition in a child,[8] reported the case of a one-year-old negress who had a liver abscess following dysentery. The abscess was confirmed at autopsy though the etiology was then unknown. Almost a century later Niblock reported the case of a Hindu boy of eleven months who, following an attack of dysentery, developed a liver abscess from which eighteen ounces of typical chocolate-colored "liver pus" was drained at operation. The pus was studied neither culturally nor microscopically, but its appearance was described as being quite typical of that obtained from patients with "tropical" liver abscess. Le Roy des Barres reported the case of an eight-month-old infant who had an abscess of the liver. There was no previous history of dysentery, but living amebas were found in pus aspirated from the abscess. The patient recovered after drainage had been instituted and a course of emetine had been given hypodermically.

From the Department of Medicine, Peiping Union Medical College.

In view of the relative infrequency of amebic liver abscess in children, it seems worth while to report an additional case in the hope of further increasing the general knowledge of the condition.

CASE REPORT

J. Y. F., a Chinese female child aged five years, was admitted to the hospital on Oct. 13, 1932, complaining of fever and abdominal pain of four weeks' duration. Her family history was unimportant. Her past history revealed that she was born in Shanghai but had lived the greater part of the first three years of her life in Anhui (Central China). She had lived in Tientsin (North China) for the past two years. Throughout her life she had had occasional attacks of mild diarrhea, each attack lasting less than a week and never being associated with dysenteric stools.

In July, 1932, two and a half months prior to admission, the patient developed diarrhea with four or five loose stools each day. The stools at first contained only mucus but soon showed moderate amounts of pus and dark blood. The patient's parents did not notice that she had any fever. Ten days after the onset of the diarrhea she was given a proprietary medicine following which her stools rapidly became normal. Thereafter she was quite well until Sept. 16, 1932, four weeks before admission, when she gradually developed an intermittent fever. Her temperature seemed to be higher in the afternoons and evenings with morning remissions. Associated with the fever she developed night sweats and moderate drowsiness. Two days later, September 18, she began to complain of an intermittent, generalized pain over the upper half of the abdomen. At times this was sufficiently severe to make her double up and cry. It was not associated with the ingestion of food, defecation, or urination. At the same time she became pale, lost her appetite, and began to lose weight. The symptoms continued with increasing severity. On October 6, one week before entry, she was noted to be less drowsy but more irritable. Her abdominal pain was more severe and was localized to the right upper quadrant. Her fever continued, and she vomited on two occasions. The vomitus contained only recently ingested food and medicine. She continued to lose weight, to be feverish, and to complain of pain in the right upper quadrant of the abdomen. Four days before entry a swelling appeared over the right lower portion of the chest and right upper abdomen. This gradually increased in size and became more painful until the time of admission.

Throughout her illness her bowels moved only once in one or two days, and her stools were hard.

Physical examination showed the patient to be a slightly underdeveloped, emaciated, and critically ill child who, when disturbed, appeared to be very uncomfortable and restless. When alone, she lay quietly with her thighs flexed. Her temperature was 38° C.; respirations, 40; and pulse, 128 per minute. Her blood pressure was 100, systolic, and 70, diastolic. Her mental state was clear, and she was not cyanotic. Her skin was dry and somewhat scaly, but normally elastic. Her chest showed moderate bulging of the right lower portion most marked at the costal margin anteriorly. Her respirations were rapid and rather shallow. Her chest expansion was limited in the lower portion of the right side. Examination of the lungs showed dullness to percussion on the right side below the third intercostal space anteriorly and over the right base posteriorly. The breath sounds were suppressed over the same region. No râles were heard. It was thought that these changes were caused by an enlarged liver. The right upper and left lung fields and the heart were normal.

The abdomen was somewhat scaphoid in appearance with a swelling in the right upper quadrant which was continuous with the swelling of the chest. The superficial veins were prominent. The abdominal wall moved slightly with respirations. The lower edge of the liver was palpated 8 cm. below the costal margin in the midclavicular line; the upper border was located by percussion in the third right intercostal space anteriorly. On palpation the liver felt smooth in outline and firm in consistency. It was markedly tender, the tenderness being most extreme over the area of greatest swelling. At this point a friction could be palpated easily, and a loud friction rub was audible on auscultation. The spleen was not felt.

There was moderate pitting edema of the feet, ankles, and lumbar region of the back.

Laboratory studies on admission revealed that the patient had a slight secondary

Fig. 1.—Roentgenogram of the chest on admission. The liver shadow is greatly enlarged with the result that the right hemidiaphragm is bulged and elevated to the sternal end of the third rib. The lung fields are slightly hazy.

anemia with 66 per cent hemoglobin (Sahli) and 4.9 million erythrocytes per cubic millimeter of blood. There were 9,450 leucocytes per cubic millimeter of blood of which 79 per cent were polymorphonuclear neutrophils; 20 per cent lymphocytes; and 1 per cent, monocytes. The urine contained a trace of albumin and occasional pus cells and hyaline and granular casts. The stools were normal and contained neither parasites nor their cysts or ova. A Mantoux test was negative. Examination of the chest by fluoroscope revealed slight haziness of the right lower lung field with a much elevated, bulging, and practically fixed right hemidiaphragm. A roentgenogram film confirmed these findings, showed that the diaphragm was at the level of the sternal end of the third rib (Fig. 1), and in addition, revealed a mild pleural thickening in the right axilla and a very large liver shadow.

A liver puncture, made immediately after admission, yielded 450 c.c. of a thick,

brownish, yellow pus with a slightly fishlike odor. Fresh smears of this pus contained from five to ten motile trophozoites of *Endameba histolytica* per high power field, but on ordinary culture media it yielded no growth. Following the liver puncture the patient felt much better. She was given general supportive treatment. On the day of admission 0.03 gm. of emetine hydrochloride was given subcutaneously. Thereafter she had 0.01 gm. of the drug, given by the same route, twice a day until she had received a total dose of 0.18 gm. She ran a moderate fever with daily rises to 38° or 39° C. On the fifth day in the hospital, Oct. 17, 1932, a second liver puncture was made. At that time 250 c.c. of pus which was quite similar in character to that previously aspirated was obtained. It again showed the motile trophozoites of *Endameba histolytica*. Thereafter her condition improved steadily. Her liver diminished in size, and on October 25 it was impossible to obtain pus when

Fig. 2.—Roentgenogram of the chest after treatment. The liver shadow is essentially normal in size. The right hemidiaphragm is slightly elevated. The lung fields are still somewhat hazy.

a third attempt was made to aspirate the abscess. Her urine cleared, she lost her edema and by the beginning of the third week in the hospital her rectal temperature ranged from 37° to 37.8° C. daily. This low-grade fever continued, however, and there was a continued rise of blood leucocytes to approximately 20,000 per cubic millimeter. There was no significant change in the differential count. There was slight persistent enlargement of the liver. Consequently a second course of emetine hydrochloride, 0.02 gm. a day by subcutaneous injection, was started on November 7 and was continued for ten days. During this time her liver diminished in size until it was no longer palpable; and her blood picture, with the exception of a slight persistent polymorphonuclear leucocytosis, returned to normal. A roentgenogram of the chest on November 17 showed slight exaggeration of the lung markings at the right base with only slight elevation of the right side of the diaphragm. The

liver shadow was normal in size, and there was no further evidence of pleural thickening (Fig. 2).

Thereafter the patient continued to improve in every respect. She was discharged on Nov. 22, 1932, greatly improved. During her stay in the hospital frequently repeated search had failed to reveal either cysts or trophozoites of *Endameba histolytica* in her stools. Nevertheless, as a precautionary measure, she was given yatren orally twice a day for two weeks after her discharge. Unfortunately her parents did not return to the hospital as requested for this treatment, and the exact dosage of the drug used is not known.

She was again seen on Feb. 25, 1933. At that time she appeared to be entirely normal. She had had no fever, abdominal pain, or other symptoms of dysentery or liver abscess since discharge, and had gained remarkably in weight. Her liver was barely palpable as a smooth edge 1.5 cm. below the costal margin in the midclavicular line. Her spleen still was not palpable, and there was no other abnormality on examination. In answer to further queries, her father wrote on Oct. 23, 1933, that the child was still in the best of health with no recurrence of symptoms.

COMMENT

The diagnosis of this child's condition offered little difficulty. With the fairly suggestive history and typical physical findings, an exploratory liver puncture was clearly indicated. This was made, as has been advised repeatedly, over the point of maximum swelling and tenderness. The finding of *E. histolytica*, which were much more numerous than is usual although the pus which was obtained was brownish yellow and not "chocolate" colored as is usually described, established the diagnosis beyond question. The absence of parasites from smears of the pus would not have altered the diagnosis as their demonstration is frequently extremely difficult or impossible. Since amebas most often occur in the the walls of the abscess, if emetine is withheld, they may be found more readily in the freshly exuded serum and pus obtained from a second or third aspiration.

The treatment of this patient was according to the usual procedure in uncomplicated cases of amebic liver abscess in this clinic.[3] Fry[11] and others have reported patients in whom large liver abscesses of undoubted amebic origin have been resorbed without aspiration or drainage, their sole specific treatment having been the subcutaneous injection of emetine. However, as has been emphasized by Le Roy des Barres,[10] infants and small children tolerate this drug poorly, especially when they are very sick. Our patient was in such critical condition on admission that we felt it unwise to rely on emetine alone. Following aspiration of the abscess, she improved so rapidly that, with bacteriologically sterile pus, there was never a question of open drainage. This latter procedure, while formerly widely used, is now, in the words of Thurston, "to all intents and purposes obsolete except under unusual conditions."[6] Because of its higher mortality rate, more difficult aftercare, greater incidence of sepsis, and longer course in the hospital, open operation now is used by most writers only in the presence of

established sepsis.[1, 2, 3, 4, 7] In addition Thurston advises open exploration with aspiration or careful cleaning of the abscess followed by tight closure of the wound and subcutaneous injection of emetine in cases in which the diagnosis is obscure or in which the signs of localization are so few or so abnormally placed as to render simple aspiration inadvisable.

The number of aspirations necessary in any given case may vary considerably. It is the practice in this clinic to repeat the procedure only when the clinical condition of the patient indicates the necessity of further evacuation of the abscess. At each time, the abscess cavity is emptied as completely as is possible. The first course of emetine given here usually covers a period of from seven to ten days. This also may be repeated if the clinical condition of the patient makes it seem advisable. As the demonstration of amebas or their cysts in the stools is frequently very difficult, it is the practice here to give each patient a course of yatren or other appropriate drug to relieve chronic intestinal amebiasis if it should be present. This is given after the liver abscess has subsided even though the stools have been found to contain no amebas.

SUMMARY

A case of amebic abscess of the liver occurring in a Chinese girl, aged five years, is reported. Aspiration of the abscess combined with subcutaneous injection of emetine hydrochloride, the treatment of choice for such a patient, was followed by apparent cure.

REFERENCES

1. Rogers, L.: Lancet **1**: 463, 569, and 677, 1922.
2. Ludlow, A. I.: China M. J. **40**: 1165, 1926.
3. Chen, S. M., Van Gorder, G. W., and Yuan, Y. K.: Nat. M. J. China **17**: 393, 1931.
4. Manson-Bahr, P.: Proc. Roy. Soc. Med. **25**: 233 (Part I), 1931-32. (Sect. Trop. Dis. & Parasitol. **25**: 1, 1931.)
5. Legrand, H.: Arch. de Méd. d. enf. **9**: 129, 1906.
6. Thurston, E. O.: Lancet **2**: 1008, 1924.
7. Murray, F. J., and Koh, P. K.: Canad. M. A. J. **26**: 312, 1932.
8. Brown: Trans. Med. Soc., Edinburgh, 1824 (quoted from Legrand[5]).
9. Niblock, W. J.: Indian M. Gaz. **46**: 137, 1911.
10. Le Roy des Barres: Rev. méd.-chir. d. mal. du foie **5**: 42, 1930.
11. Fry, A. S.: Indian M. Gaz. **59**: 488, 1924.

GANGRENE OF AN EXTREMITY IN A NEWBORN INFANT

With a Review of the Literature

F. Curtis Dohan, M.D.
Merion, Pa.

BECAUSE of the rarity of reported cases of gangrene of the extremities in the newborn, and because of the need for further study of the origin of this condition, the following case is presented:

On April 1, 1933, a nineteen-day-old white boy, with a gangrenous left foot, was admitted to the service of Dr. Joseph Stokes, Jr., in the Hospital of the University of Pennsylvania. The history to the date of admission was obtained from the family doctor, who delivered the child, as well as from the parents and maternal grandmother.

The mother, an apparently normal and healthy young primipara, was delivered March 12 of her "eleven-pound" son after three hours of labor. It was a right occiput anterior presentation. The umbilical cord which was around the infant's neck was slipped over the head before delivery of the body. No amniotic bands were noted. There was no known traumatic incident that might have affected the infant at any time before, during, or after delivery. Morphine was the only drug administered before the birth of the patient. Except for some cyanosis immediately after birth, no abnormalities were noted until "the second or third day," when the patient exhibited a diffuse skin eruption. Irritability, anorexia, and vomiting soon became prominent features which did not respond to diluted milk formulas. The child would not nurse at his mother's breast. The malnutrition increased. At times the baby "felt warm," but no temperature record was kept. On the thirteenth postnatal day the grandmother noticed that the left foot was cold and that there were a few pinhead-sized "blue spots" upon it. By the seventeenth day the foot was frankly gangrenous and the patient was brought to the hospital two days later.

At this time the foot was a dusky blue red, with a swollen dorsum. There was a small erosion on the dorsum and another just above the internal malleolus, also, a distinct, irregular, raised line of demarcation about an inch above the malleoli, and extending entirely around the ankle. Arterial palpation was unsatisfactory. The skin lesions were mostly small erythematous macules and papules with many vesicles and fewer pustules. They almost completely covered the infant with the exception of the soles of the feet. There were some erosions on the palms of both hands. The dermatologic lesions were distributed chiefly over the scalp, face, and upper thorax. The infant was not weighed on admission because of the temporary feeling that the disease might have been infectious.

The general nutritional status of the patient was poor. There was moderate dehydration; rectal temperature was 100.2° F. The umbilical stump was not completely healed, and the umbilical depression contained a small amount of yellow, mucouslike substance which appeared to be the remains of Wharton's jelly. Besides the above, no other abnormalities were noted. The heart was studied with particular care since a cardiac abnormality had been suggested; the rate was around 170 per minute.

From the Pediatric Service of the Hospital of the University of Pennsylvania and the Pediatric Department of the School of Medicine of the University of Pennsylvania.

April 3, two days after admission, Dr. E. L. Eliason and Dr. C. W. McLaughlin, performed a guillotine amputation in the middle third of the left lower leg, under local anesthesia. No tourniquet was used and "both the anterior and posterior tibial arteries were seen to be completely thrombosed at the site of the amputation."

Under a regime of calamine lotion for the skin, and an ointment of ammoniated mercury for the scalp, the dermatologic aspects soon greatly improved. A simple formula of evaporated milk, corn syrup, and water was well taken by the child so that his malnutrition and dehydration quickly disappeared and a satisfactory weight gain was established. Convalescence was essentially uneventful. Two occasions of moderate fever were attributed to mild upper respiratory infections. The temperature was at other times within normal limits. The infant was discharged to the care of the family physician on April 27, and two months later he reported that the child was in perfect condition and the stump well healed. The infant's weight on discharge was 9 pounds 1½ ounces.

Laboratory studies were not helpful in determining the etiology of the case. Roentgenogram showed that "the left leg is negative for any evidence of fracture. The appearance of the bones is suggestive of congenital lues." But evidence of syphilis was lacking in the parents. The father's blood serum was negative (Kahn test only) as was the mother's (repeated Kahn and Wassermann tests). The infant's Wassermann reaction was reported moderately positive and the Kahn test as weakly positive, but a blood sample taken three days later was negative. Later evidence has also confirmed the fact that the child has not suffered from syphilis. Blood counts, save for evidence of dehydration on admission, were about the same. Thus, on April 4, the red cells were 5,800,000 per cubic millimeter; white cells 18,500 per cubic millimeter, of which 70 per cent were large mononuclears; the platelet count was 228,000 per cubic millimeter; 7 per cent of the red cells was reticulated. On April 24 the blood coagulation time, by a modification of the Lee and White technic, was five minutes and the clot retraction "was good." On the same date, three hours after a feeding, the blood sugar was 66 mg. per cent. Urinalysis, except for some albumin on a few occasions, was negative. A blood culture taken on April 7 showed no growth "except a few *Staphylococcus albus* from the deep plate." These were regarded as contaminants. Histamine tests for circulatory capacity as adapted by Starr from Lewis' technic, were made April 8 because the right foot was quite cold, and it was feared that it, too, might become gangrenous. Compared with another infant of about the same age and with adult standards, the patient showed moderately subnormal reaction to histamine on the dorsum of the right foot. On April 24, three days before discharge, this test gave a normal reaction.

The pathologic report submitted by Dr. Albert Bothe, concerning the amputated foot follows. "Gross examination: Specimen consists of foot which is cyanotic from just above the ankle. There is one ulceration just above the internal malleolus and also on the dorsum. Sections were made from the anterior and posterior arteries.

"Microscopic examination: There is no evidence of obliteration of the lumen. The media is edematous and shows inflammatory changes of the chronic type.

"Diagnosis: mediitis of the anterior and posterior tibial arteries."

What became of the thrombi observed at amputation is not known. A culture taken from the proximal end of amputated portion of the leg "reveals no growth except anaerobic gram-negative spore-bearing rod, probably contaminant."

LITERATURE

Over one hundred years ago Martin[1] described bilateral lower leg gangrene which commenced as black flecks just above the feet. The

child enjoyed an easy birth from a thirty-three-year-old "hysterical" woman who six months previously had suffered ptyalism following vigorous calomel therapy for a "liver inflammation." Neonatal inspection of the child revealed a healthy, although somewhat small, infant, with a hematoma over the left parietal bone. No other abnormalities were noted until the eighth extrauterine day when evidences of gangrene made their appearance. The child died on the twenty-fourth day.

Eleven years later, in 1839, Richter[2] reported a patient seen by him with numerous gangrenous spots on the extremities; another case was reported anonymously of a male infant who was born with dry gangrene of all parts below the hypogastrium. This medical curiosity is said to have lived ten extrauterine hours.

At a meeting of the Gesellschaft für Geburtshülfe in Leipzig, February 21, 1876, three members told of cases of gangrene in the newborn. Leopold[3] reported a child (born of a para iv mother, with a rachitic pelvis), who developed gangrenous feet fifteen days after delivery by version and extraction with the right foot. On the eighteenth day the patient died, showing signs of pneumonia as well as gangrene of the left foot and a portion of the leg, and less marked gangrene of the right. The autopsy showed pneumonia of both lower lobes and a patent foramen ovale. There was a thrombus extending from the lower third of the abdominal aorta part way down the femoral arteries. The neighboring great veins did not contain thrombi.

Ahlfeld[3] next described a case detailed to him by the infant's mother. The labor was long, and the child was somewhat asphyxiated at birth. On the ninth postnatal day there appeared on the lateral side of the right calf a fair sized blue mark. Camphor splints and bandages were applied. Two days later the leg was black, with a blister at the site of the original blue spot. The leg was amputated, the child dying the following day.

Weichert[3] told of a poorly remembered case in which ten days after birth both lower legs were gangrenous. Pus was found in the umbilical vein. This was thought to be the origin of an embolus.

A somewhat similar case was described by Wieland[4] in 1912. At the end of the second week of infancy, a firstborn child of healthy parentage developed an apparent inflammation of the distal segment of two fingers of the right hand, associated with the discharge of a cloudy fluid from the navel. Both lesions seemed to clear up under treatment, but five weeks later the finger tips had again become swollen and a line of demarcation was evident. Somewhat later these gangrenous tips sloughed away. The child recovered. The pathologic report was not noteworthy.

Obadalek[5] reported an infant who developed dark red flecks upon its left heel fourteen days after an easy birth. By the end of the third week the line of demarcation was just under the knee. Amputation was per-

formed in the fifth week; at operation it was noted that the anterior tibial artery was patent. The pathologic examination of the amputated portion showed many white blood cells in the dilated vessels. Although the intima was unchanged, the media in many places was edematous.

Grassi[6] in 1929 described a case seen by him and reviews the literature. The patient was the second child of a thirty-five-year-old mother who had had malaria a few years before, as had the father. The delivery was spontaneous. The presentation was left occiput anterior. The umbilical cord was around the infant's neck, and therefore it was somewhat cyanotic and asphyxiated; artificial respiration was employed by the midwife in attendance. On the third day a rosy discoloration of the right forearm was noted. Dry gangrene, plus secondary infection, was present on the tenth day when the patient was seen by a physician. The arm was wrapped in dressings saturated with absolute alcohol. Auto-amputation occurred on the thirty-seventh day followed by recovery. The parents' blood Wassermann reactions were negative. No pathologic report was given.

Corret, Levy, and Beau[7] tell of an infant who showed frank gangrene of a lower leg on the twelfth postnatal day. Amputation was successful. The mother had a thyroidectomy twenty years before and was delivered of a macerated twin one year before the birth of the patient. The mother's blood Wassermann reaction was negative and the father "denied all syphilis." A microscopic examination of the amputated portion showed a periphlebitis and periarteritis associated with an endophlebitis. The arteries contained organizing thrombi.

A possible pathogenesis is suggested in the case published by W. Fischer.[8] The midwife who attended the eighteen-year-old primiparous mother stated that after fourteen and a half hours of hard labor the infant was born with the palm of his right hand over his right eye, and the umbilical cord around his neck. About one week after birth the right hand appeared bluish and was frankly gangrenous, as was the forearm on the eleventh day when he was brought to the hospital. Amputation was deemed unwise because of the patient's poor physical condition. The child died on the fourteenth day. The blood Wassermann reaction was negative, and the blood culture showed a growth of proteus. The postmortem examination showed thrombus formation in the great vessels of the right arm, lung arteries, and aorta, while in some places it showed beginning organization. "Long bacilli" were demonstrated in some portions of the thrombi.

Among the few case reports in English are the following: Cotes-Preedy[9] writes of an infant whose multiparous mother had fallen twice on her right side before delivery, but she delivered spontaneously. The presentation was "right occiput anterior." The cord was twice around the baby's neck. It was especially noted that there were not any

amniotic bands around the right foot of the infant. Gangrene soon became apparent, and the child died on the fourteenth postnatal day. No vessel block was found.

Fitzwilliams and Vincent[10] describe the misfortune of an infant who had a normal birth from an apparently healthy mother. The lateral portion of the lower right leg showed signs of gangrene on the tenth day, and by the fourteenth day the line of demarcation was evident. On the twenty-fourth day a pustulous eruption appeared. The parents at last consented to amputation, which was done, and the child recovered. The parents' blood serum was negative for evidence of syphilis. An examination of the amputated portion showed slight thickening of the endothelium of the anterior tibial artery.

Löwy[11] has reported gangrene appearing in the distal portions of some of the toes of a healthy infant five days after delivery from an apparently healthy mother. Foltanek[12] mentioned a case of gangrene of the foot appearing on the eighth postnatal day. Haverschmidt[13] tells of a suckling with a lumbar meningomyelocele and symmetrical gangrene of the soles of the feet. They were healed after a successful plastic operation upon the meningomyelocele. He also mentions another case of gangrene in an extremity, caused, he thought, by embolus from an infected navel. In Freund's[14] case the infant was a frontal presentation. It was noticed after his delivery from a "strong woman" that the left hand was cold and anemic and that there was a furrow around the forearm above the wrist. On the eighth day the hand was gangrenous. Two days later the child died with symptoms of pneumonia.

De Bruin[15] reports an infant who had a gangrenous right leg at birth which shed spontaneously in a few weeks. The mother had had a previous miscarriage (at six months). Delivery was spontaneous two days after rupture of the amniotic sac. Forceps application and version had previously been attempted with an incompletely dilated cervix.

A very interesting case is described by Barmwater.[16] To a mother with asthma and heart disease there was born her second child. It was cyanosed and showed blisters on the extremities and around the mouth and lips. The child died on the ninth day, after the extremities had become gangrenous. The autopsy protocol noted that the heart was enlarged, the septum atriorum defective, and the ductus arteriosus patent. There was slight stenosis of the isthmus aortae. The arteries and veins of the right leg were normal. There was evidence of gangrene of both hands and the lower parts of both legs. The author stated that a spasm of the vessels similar to that in Raynaud's disease was the most likely cause.

Durando Durante[17] has published a report concerning two patients, seventeen and twenty-four days old, who died a few days after onset of gangrene. In one the process was more or less confined to the feet,

while in the other the lower abdomen and upper legs were the seat of the gangrene. Castana[18] published a case in which postnatal cyanosis seemed to develop into gangrene in some of the toes and the whole lower left arm and hand. Bronson[19] has reported an infant whose spontaneous birth from an apparently healthy mother was abnormal in that the umbilical cord was wrapped around its neck. On the first day it was noticed that there were gangrenous areas on the right cheek, neck, ear, clavicle, and right thumb. This was quite superficial, but part of the ear sloughed away. The doctor who had first seen the case had treated it with a solution of lysol.

A case of skin gangrene of the dorsum of the heel and part just below the internal malleolus of the right foot is reported by De Snoo.[20] Following delivery with forceps the child was given artificial respiration because of poor breathing. The gangrene eventually disappeared.

DISCUSSION

Analysis of the data fails to reveal any one significant etiologic factor. One might consider as causes, angiospasm in seven cases, traumatic delivery in three cases (a factor that those in attendance might minimize), infected emboli in three, and amniotic bands in one, but due to the insufficiency of the data or to their completely negative character, one would find it difficult to discover etiologic explanations for the other cases. There is one common denominator, and that is the time of onset. Thus, four showed gangrenous signs of birth, three in more than eighteen days, but thirteen cases showed gangrenous development within fifteen days and of these most were from eight to twelve days. This may mean that in these cases the etiologic factors were operative at similar times, more probably during labor.

Despite the fact that the data on the case reported above appear to be more complete than any of the cases reviewed, the etiology is not clear.

SUMMARY

A case is reported of gangrene of the left lower leg and foot, which appeared about the thirteenth postnatal day. Blood serologic tests for syphilis were negative on the parents. The infant's blood serologic tests for syphilis were negative on repetition, following a moderately positive reaction in the first blood sample. A roentgenogram examination of the legs was negative for bony displacement but indicative of congenital syphilis. Histamine tests showed a circulatory efficiency of the non-gangrenous leg which was distinctly below normal. Blood sugar, blood coagulation, blood counts, and urinalysis, as well as body temperature and other clinical observations, deviated so slightly from the normal that they were not considered as positive evidence to the solution of this unsolved problem. Pathologic and bacteriologic studies were inconclusive.

A survey of the literature was made in which twenty-five reported cases were reviewed. Analysis of the data reported suggested that the etiologic factors were at present obscure. Trauma, infected or non-infected embolism, constricting amniotic bands, and angiospasm, may all play a part.

The author wishes to express his gratitude to Dr. Joseph Stokes, Jr., for his encouragement and suggestions in preparing this report.

REFERENCES

1. Martin: Mag. f. d. ges. Heil (Berlin) 27: 428, 1828.
2. Richter: L'Expérience (Paris) 2: 193, 1839.
3. Leopold, Ahlfeld, and Weichert: Arch. f. Gynäk. 10: 189, 1876.
4. Wieland: Deutsche Ztschr. f. Chir. 116: 783, 1912.
5. Obadalek, W.: Arch. f. Kinderh. 90: 111, 1930.
6. Grassi, A.: Riv. di clin. pediat. 29: 120, 1931.
7. Corret, Levy, and Beau: Rev. méd. de l'est 60: 64, 1932.
8. Fischer, W.: Arch. f. Gynäk. 136: 181, 1929.
9. Cotes-Preedy: Lancet 1: 575, 1904.
10. Fitzwilliams and Vincent: Lancet 1: 753, 1913.
11. Löwy: Mitt d. Gesellsch. f. inn. Med. u. Kinderh. 21: 152, 1922.
12. Foltanek: Mitt d. Gesellsch. f. inn. Med. u. Kinderh. 5: 191, 1906.
13. Haverschmidt: Jahrb. f. Kinderh. 65: 624, 1907.
14. Freund: Verhandl. d. deutsch. Gesellsch. Gynäk. 8: 500, 1899.
15. De Bruin: Nederl. tijdschr. v. Geneesk. 2: 1349, 1906.
16. Barmwater, K.: Wien. klin. Wchnschr. 38: 220, 1925.
17. Durante: Méd. Infant, 222, 1898; cited by Barmwater.[16]
18. Castana, V.: Pediatria 31: 1305, 1923; cited by Barmwater.[16]
19. Bronson: Tr. Am. Dermat. Assn., p. 158, 1901.
20. De Snoo, K.: Nederl. tijdschr. v. Geneesk. 72: 3915, 1928.

766 Beacom Lane

RELATION OF "CORRECT" WEIGHT AND BLOOD FINDINGS TO PHYSICIANS' ESTIMATES OF NUTRITION OF SCHOOL CHILDREN

REGINE K. STIX, M.D., AND CLYDE V. KISER, PH.D.
NEW YORK, N. Y.

HOW valuable are the ordinary measures of nutrition for children of school age? Results bearing on this question from recent physical examinations of 514 school children from New York's "lower East Side" are presented in this paper. The methods employed for measuring nutrition were (1) physicians' general appraisals (good, fair, poor, or very poor) made immediately after the completion of each examination; (2) the Baldwin-Wood weight-height-age scale; (3) the Dreyer tables of "expected" weight for given chest circumference and sitting height; (4) the Tallqvist scale for determination of the hemoglobin content of the blood; and (5) the red blood cell count.

It should be emphasized at the outset that the methods used are the ordinary ones. The "correct weight" tables, particularly, are still used extensively in private practice and in schools and clinics despite previous findings regarding their limitations. For just this reason data were secured which would make possible a comparison of physicians' ratings of nutrition with the indirect and commonly used "indices."

By way of orientation a brief statement of the original purpose of the physical examinations and nature of the sample may not be amiss. The work was conducted in the spring of 1933 by the United States Public Health Service and the Milbank Memorial Fund with the assistance of the New York City Department of Health. The chief object was to ascertain the prevalence of malnutrition in children of school age in relation to family income, and as such the study was part of a more general survey of the effect of the depression on the health of marginal groups of urban population. The children selected for study were from "lower East Side" families from which income and sickness data had been secured.

As would be expected from the area, the children selected for study were largely of foreign parentage and came from families of low economic status. They were generally of South European stock with Italians forming the largest single group. Approximately 60 per cent of the children examined came from families reporting incomes amounting

From the Milbank Memorial Fund.

to less than $4.00 per capita per week; 40 per cent from families on relief, and less than one-fifth from families earning $6.00 or more per capita per week in the spring of 1933.

A little over one-third of the children were rated by examining physicians as "poor" or "very poor" in nutritional status, a proportion presumably much higher than would be expected in a nondepression era. More convincing evidence of the relation of nutrition of children to economic conditions was revealed by internal differences in the sample. Although practically all of the families belonged to the lower order of economic classes, a direct association between nutritional status and family income was manifest. Furthermore, a lowering of nutritional status appeared to be associated with drop in family income in as short a time as a year.[1] The examining physicians, of course, knew nothing of the economic background of any child examined. The economic data were secured independently by other investigators.

The number of children examined is too small to warrant sweeping conclusions, but the indications that nutrition of school children in borderline families has been impaired by the depression conform with the logic of the situation. The findings of an independent diet survey[2] of these families are consistent with those of the nutrition study. Similar nutritional studies have yielded substantially the same results.[3]

The physicians' estimates of nutrition are admittedly not infallible. They were based upon a single examination, taking into account the child's general physical condition, with special emphasis on the amount of subcutaneous fat present, quality of muscle tone, and texture of skin and hair. Another group of equally competent physicians might have appraised certain of the children differently even if the same criteria had been used. Nevertheless, it is generally admitted that physicians' estimates of nutrition must serve as the standard until a more universally accepted definition of nutrition is given and until methods are devised for measurement which are better than the present ones based largely upon "correct" weight.

There is evidence of general agreement among the three physicians making these examinations as to the clinical concept of "poor" nutrition. The children were referred to one of three physicians, as they came from their classrooms, without selection. The percentage distributions of each physician's ratings were separately computed and found to be quite similar. About 25 per cent of the children examined by each doctor were rated "good," and about 33 per cent to 38 per cent were rated "poor" or "very poor." In addition, one of the doctors saw every child, and where there was an occasional discrepancy as to findings or rating, all three physicians examined the record and agreed on the rating.

A further check may be found in the rating of brothers and sisters. There were 130 such cases involving 342 children, many of them ex-

amined at different schools and by different doctors. Although the doctors had no way of knowing whether or not the children were related, there was a marked similarity in the rating of siblings. In more than 100 out of the 132 families, the ratings for all children in the family examined were the same or only one step removed in the nutritional scale.

CORRELATION OF NUTRITION ESTIMATES WITH INDICES OF WEIGHT

While "standard" weight tables have been in general use for a number of years, there has been a growing dissatisfaction with their adequacy as indices of nutrition or physical fitness. Careful studies conducted by the United States Public Health Service[4] have indicated inadequacies of the Baldwin-Wood[5] and Dreyer[6] scales in their application to school children whose nutrition had been appraised by experienced physicians. Dublin[7] has pointed out practical difficulties involved in the use of the Dreyer scale as a standard for acceptance or rejection of candidates for life insurance. More recently Franzen[8] and Pryor and Stolz[9] have offered other measures which they believe will more adequately meet the requirements of a nutrition index.

Analyses of the data tend to confirm the results of previous studies concerning the inadequacy of the Baldwin-Wood and Dreyer scales as indices of nutrition. The proportion of children rated "underweight"* by the tables is substantially smaller than the proportion rated "poor" by the physicians. On the basis of the Baldwin-Wood scale 8 per cent of the boys and 13 per cent of the girls were "underweight." According to the Dreyer scale, 3 per cent of the boys and 28 per cent of the girls were "underweight."† The physicians appraised 40 per cent of the boys and 30 per cent of the girls as being "poor" in nutritional status. Thus, while a larger proportion of boys than girls were appraised as "poor" by physicians, the incidence of "underweight" was, according to the weight indices, greater among girls.

A cross classification of nutritional appraisals and weight indices reveals certain rough consistencies and further differences (Table I). Overweight children were generally appraised as "good" in nutrition, and pupils underweight by the Baldwin-Wood scale failed to get that appraisal. However, one-fourth of the girls of good nutritional status were underweight by the Dreyer scale. Most of the girls of that nutri-

*According to frequent usage, a child is regarded as "underweight" by the Baldwin-Wood standard if his actual weight is more than 10 per cent below the "normal" weight for children of identical sex, age, and standing height. He is "probably underweight" according to the Dreyer scale if his actual weight is more than 10 per cent below the expected for identical sex, chest circumference, and trunk length.

†The extremely marked sex difference in proportion underweight by the Dreyer scale has been encountered by other investigators[6b]. This may be due to errors in technic of securing chest measurements of adolescent girls, or it may be due to inaccurate allowance for the factor of breast development, in Dreyer tables for expected weights for girls of given chest circumference.

TABLE I

DISTRIBUTION BY WEIGHT STATUS ACCORDING TO THE BALDWIN-WOOD AND DREYER INDICES OF CHILDREN WITH SPECIFIED NUTRITION APPRAISALS

PHYSICIANS' ESTIMATES OF NUTRITION	BALDWIN-WOOD SCALE				DREYER SCALE			
	NUMBER CHILDREN INVOLVED*	PER CENT			NUMBER CHILDREN INVOLVED†	PER CENT		
		UNDER-WEIGHT 10+ PER CENT	NORMAL WEIGHT	OVERWEIGHT 20+ PER CENT		UNDER-WEIGHT 10+ PER CENT	NORMAL WEIGHT	OVERWEIGHT 15+ PER CENT
Boys								
Good	41	0.0	73.2	26.8	36	0.0	86.1	13.9
Fair	110	1.8	95.5	2.7	101	1.0	96.0	3.0
Poor	101	17.8	82.2	0.0	92	5.4	94.6	0.0
Girls								
Good	84	0.0	65.5	34.5	80	25.0	71.3	3.7
Fair	98	12.2	85.7	2.0	95	26.3	73.7	0.0
Poor	75	28.0	72.0	0.0	72	31.9	66.7	1.4

*Measurements for Baldwin-Wood Scale unknown—5 girls.
†Measurements for Dreyer Scale unknown—23 boys and 15 girls.

tional and weight status were over twelve years of age—a confirmation of the inadequacy of the Dreyer scale applied to adolescent girls.*

The main inadequacy of weight tables is probably exemplified best by the figures which indicate that the "poor" nutritional ratings are not confined to underweight children. Only 22 per cent of the children appraised as "poor" in nutrition were underweight by the Baldwin-Wood scale, and only 17 per cent were underweight by the Dreyer scale.

Although the indices of weight identify only a small proportion of the children of poor nutritional status, the question may still be raised concerning the possible use of these indices to ascertain the relative proportion of underweight children in different groups. Do differences in the proportion of underweight afford an indication of whether children in one school, one neighborhood, or one economic group are better or worse in nutrition than those in another? The observed discrepancy in the order of the sexes with respect to proportions appraised as poor in nutrition and proportions "underweight" would seem to constitute a serious inadequacy of the weight indices for use even in this limited capacity. As a further test, however, the children were divided into four groups on the basis of economic condition of families from which they came, and the order of the groups with respect to incidence of underweight was compared with that with respect to proportion of children appraised as "poor" in nutritional status. A rough similarity appears, in that the lowest income groups had higher proportions of children "underweight" and "poor" in nutritional status than did the families at the upper end of the economic scale (Table II). However, the inverse correlation between economic status and good nutrition was more consistent than that between economic status and "correct" weight. However, these comparisons are based on a small number of cases and can be no more than suggestive in their implications.

CORRELATION OF BLOOD TESTS WITH PHYSICIANS' APPRAISALS OF NUTRITION

There is a dearth of literature concerning the relation of the blood picture to nutritional status among "normal" children. For that reason, results concerning this point which were secured in the course of examination of our group of New York school children are here presented.

Two blood tests were made on each child examined: an estimation of the hemoglobin content of the blood by the Tallqvist method, and a red blood count. The tests were made by two technicians, lent by the United States Public Health Service for the study. Laboratory conditions, however, were far from ideal, and the speed with which the tests had to be made (on some days as many as forty-five or fifty of each type per day) may to some extent have affected the reliability of the data obtained. The correlations between hemoglobin estimations by the Tallqvist scale

*This scale is partly dependent on the accurate measurement of chest circumference, which is difficult to obtain when no satisfactory allowance is made for breast development.

TABLE II

PERCENTAGE OF CHILDREN FROM FAMILIES OF SPECIFIED ECONOMIC STATUS WHO WERE APPRAISED "POOR" IN NUTRITION AND PERCENTAGE "UNDERWEIGHT" BY BALDWIN-WOOD AND DREYER INDICES

	PER CENT "POOR NUTRITION" OR "UNDERWEIGHT"			
	A*	B*	C*	D*
	MISC. RELIEF	HOME RELIEF	NON-RELIEF UNDER $4.00 PCWI	NON-RELIEF $4.00 AND OVER
Appraised "poor" by physicians				
Both sexes	40.4	37.2	41.4	27.8
Boys	50.0	43.8	38.2	34.8
Girls	28.6	30.6	44.6	21.7
"Underweight" more than 10 per cent				
Baldwin-Wood				
Both sexes	11.9	11.1	11.9	8.6
Boys	9.4	11.0	5.5	6.5
Girls	14.8	11.3	18.5	10.5
Dreyer				
Both sexes	17.2	19.8	19.4	9.8
Boys	6.5	1.6	5.9	0.0
Girls	29.6	36.8	32.7	18.0
Total number children involved				
Both sexes	60	145	111	198
Boys	32	73	55	92
Girls	28	72	56	106

*A—Miscellaneous Relief Class—chiefly work relief families.

B—Home Relief—families receiving regular food allowances from Home Relief Bureau. The diet survey indicated that the food secured by these families was more adequate than that secured by families in Class A or C.

C—Non-Relief—family income less than $4.00 per capita per week in 1933.

D—Non-Relief—family income $4.00 or more per capita per week—presumably a class less affected by the depression than any of the other three.

and red blood counts showed such a large proportion of children with a color index over 1.0 that we were forced to assume that either the hemoglobin estimations or the red blood counts were subject to wide error. Since the Tallqvist scale is recognized to be inadequate as compared with more accurate hemoglobinometers, and since the conditions under which these tests were made admitted of wide error in reading the scale due to inadequate lighting, drying of some specimens and fatigue on the part of the technicians, we assumed that the red counts, though subject to some error, were the more reliable test. The hemoglobin estimations were not used.

Little variation was manifested in the distribution of the red cell count, whether the children were rated "good," "fair," or "poor" in nutritional status. We have arbitrarily chosen from 3,700,000 to 4,500,000 as our middle range because our median count was about 4,100,000. Approximately one-half of the pupils, regardless of nutritional ratings, came within this range. However, a little over one-third of the pupils appraised as "good" in nutritional status as compared with less than one-fourth of those rated as "poor" had red cell counts of more than 4,500,000 (Table III). The correlation was more marked

TABLE III

CORRELATION OF RED BLOOD CELL COUNT WITH PHYSICIANS' ESTIMATES OF NUTRITION

RED BLOOD CELL COUNT	NUTRITION APPRAISALS					
	GOOD		FAIR		POOR AND VERY POOR	
	NO.	PER CENT	NO.	PER CENT	NO.	PER CENT
*Both Sexes**						
Total	116	100.0	204	99.9	172	100.0
Under 3,700,000 per c.mm.	18	15.5	57	27.9	40	23.3
3,700,000 to 4,499,999	58	50.0	97	47.5	91	52.9
4,500,000 and over	40	34.5	50	24.5	41	23.8
Boys						
Total	41	100.0	110	100.0	97	100.0
Under 3,700,000	6	14.6	30	27.3	27	27.8
3,700,000 to 4,499,999	17	41.5	54	49.1	51	52.6
4,500,000 and over	18	43.9	26	23.6	19	19.6
Girls						
Total	75	100.0	94	99.9	75	99.9
Under 3,700,000	12	16.0	27	28.7	13	17.3
3,700,000 to 4,499,999	41	54.7	43	45.7	40	53.3
4,500,000 and over	22	29.3	24	25.5	22	29.3

*R.B.C. unknown—4 boys and 18 girls.

for boys than for girls; thus 44 per cent of the boys rated "good," had red blood counts of more than 4,500,000 while only 29 per cent of the girls rated "good," fell into this classification. Conversely, 28 per cent of the boys rated "poor" or "very poor" had red blood counts below 3,700,000 while only 17 per cent of the girls so rated had blood counts below this figure. Whether this sex difference is real or is simply a chance variation must await further investigations of larger numbers of children.

SUMMARY

Following the physical examinations of 514 school children from New York's "lower East Side," physicians' estimates of nutrition were compared with the Baldwin-Wood and Dreyer indices of "correct" weight and with red blood counts. Relative to these comparisons the following may be said:

1. Weight tables are not sufficiently accurate for a satisfactory diagnosis of nutrition of school children. Extreme deviations from average weight seem to be important factors in physicians' judgments, but weight is by no means the sole criterion of the clinical concept of poor nutrition. The seriously "underweight" pupils did not receive "good" nutritional ratings while the "overweight" pupils did. The "normal" weight pupils, however, in the nature of the case constitute the bulk of the students, and these exhibit a high degree of variability in nutrition ratings. Weight indices must be far more refined for detection of the early conditions of malnutrition.

2. The data indicated only slight correlation between blood picture and nutritional status among children of school age.

REFERENCES

1. Kiser, Clyde V., and Stix, Regine K.: The Milbank Memorial Fund Quarterly Bull. 11: 299, 1933.
2. Wiehl, Dorothy G.: The Milbank Memorial Fund Quarterly Bull. 11: 308, 1933.
3. Child Welfare News Summary, July 12, 1933, Children's Bureau, United States Department of Labor.
4. (a) Clark, T.; Sydenstricker, E.; and Collins, S. D.: Washington, U. S. P. H. S., Public Health Rep. 38: 39, 1923.
   (b) Clark, T.; Sydenstricker, E.; and Collins, S. D.: Pub. Health Rep. 38: 1239, 1923.
   (c) Clark, T.; Sydenstricker, E.; and Collins, S. D.: Pub. Health Rep. 39: 518, 1924.
5. Baldwin, Bird T., and Wood, T. D.: Weight-Height Age Tables for Boys and Girls of School Age, Supplement to Mother and Child, July, 1923.
6. Dreyer, Georges, and Hanson, G. F.: The Assessment of Physical Fitness, New York, N. Y., 1921, Paul B. Hoeber.
7. Dublin, Louis I.: The Work of Dreyer in Relation to Life Insurance Examination, Metropolitan Life Insurance Company, New York, 1922, 16 pp.
8. (a) Franzen, Raymond: Physical Measures of Growth and Nutrition, New York, American Child Health Association, 1929.
   (b) Palmer, George Truman: Child Health Bulletin, March, 1930.
   (c) Whitney, Anne: Child Health Bulletin, March, 1930.
9. Pryor, Helen B., and Stolz, Herbert R., J. PEDIAT. 3: 608, 1933.

THE PROBLEM OF FREE DIPHTHERIA IMMUNIZATION IN THE SCHOOL

EDWIN HENRY SCHORER, M.D., AND MORRIS POLSKY, M.D.
KANSAS CITY, MO.

FOR several years a number of organizations in Kansas City have taken it upon themselves to urge free immunization against diphtheria. Since 1923 such free immunization has been practiced city-wide in the public and parochial schools; parents of children of school age and even of preschool age have been urged and persuaded to bring their children to the school for such immunization. The school nurses have been active in this work.

Various schemes were tried, but usually the teachers in the lower grades had the pupils in the penmanship class work out as an exercise a letter to their parents telling them that on a certain date, at a specified time, the Board of Health would give free diphtheria immunization to the children in school. In other classes letters were written by the pupils asking permission to be given the immunization on a certain date. This propaganda was furthered by the Health Department, the Health Department nurses giving the injections and the materials used being supplied by that department.

The extent to which this immunization progressed in the schools is shown by the report (Table I) issued by Dr. Calvin Cooper, then Director of Health, in the fall of 1932 on immunizations given at the public schools.

TABLE I

YEAR	ENROLLMENT	T-A-T MIXTURE	PERCENTAGE	MISSED	PERCENTAGE
1930	43,519	32,219	74.0	11,301	26.0
1931	43,604	34,686	79.5	8,918	20.5
1932	43,016	36,718	85.4	6,208	14.5

From such wholesale immunization it was to be expected that a marked decrease in the incidence of diphtheria in the city would take place. That this happened is evidenced by the Health Department's statistics. In the four years before free school immunization was begun (1920 to 1923, inclusive), there were 2,891 cases of diphtheria with 226 deaths, or an average of 723 cases with 56.5 deaths per year; while in the last four years of such free school immunizations (1929 to 1933, inclusive), there were 964 cases with 75 deaths, or an average of 193 cases with 15 deaths yearly.

From the Pediatric Service of the Kansas City General Hospital.

When we refer to the records of the Isolation Division of the Kansas City General Hospital, a charity institution, it is rather surprising to note that in the four-year period, 1929 to 1933, inclusive, fifty-two more children with diphtheria were admitted to the hospital than in

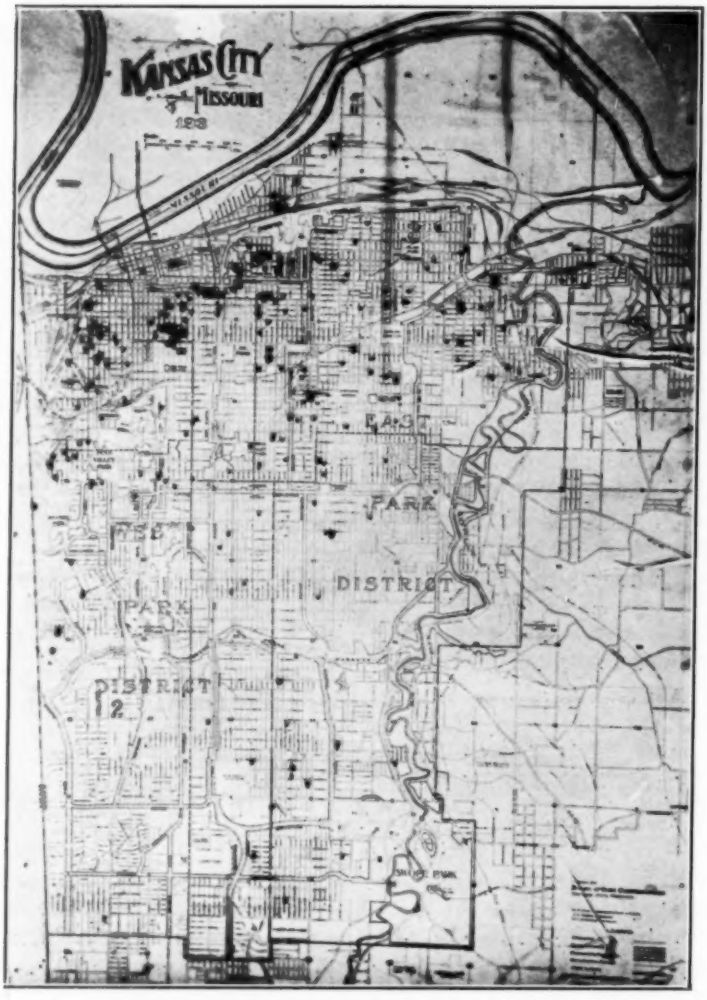


Fig. 1.—Spot map showing geographic distribution of children admitted to the hospital because of diphtheria from 1929 to 1933 (inclusive).

the four-year period, 1919 to 1923, inclusive, before immunization was begun. Furthermore, comparing the total number of cases of diphtheria and those hospitalized at the Isolation Division, we find that from 1919 to 1924, 15.46 per cent of the diphtheria patients were hos-

pitalized in the free Isolation Division; while from 1929 to 1933, 52.28 per cent were admitted.

On examining the spot map representing all the cases in children hospitalized in the years 1929 to 1933, we find that nearly all of the children came from the districts in which the less fortunate financially reside.

It is reasonable to assume that the spot map (Fig. 1) is a fair repre-

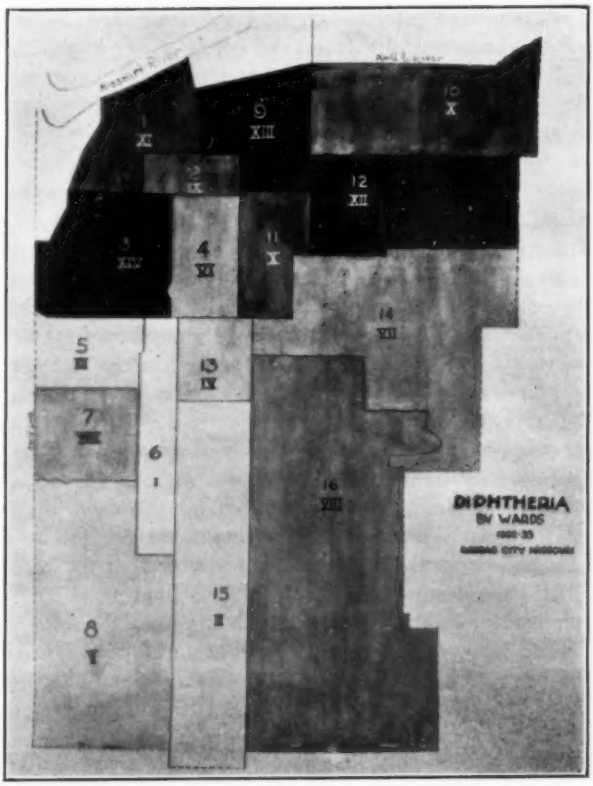


Fig. 2.—Map showing geographic distribution according to wards of all cases of diphtheria occurring in Kansas City in 1932 and 1933. (Arabic numbers designate wards, and Roman numbers, rating as to incidence of diphtheria.)

sentation of all the cases occurring in Kansas City, as illustrated by the relative incidence in the ward map shown (Fig. 2).

Fig. 2 represents all of the cases occurring in Kansas City during 1932 and 1933, the only years for which ward figures are available. It was noted that in the best residential districts there were very few cases. In this connection particular attention is called to the fact that in the past five years the entire number of diphtheria patients seen by

the pediatrists in Kansas City in private practice aggregates less than thirty.

One asks what proportion of the children admitted to the isolation ward because of diphtheria had been immunized. We tried to ascertain this, but neither the children nor their parents had reliable knowledge on the subject; some children were supposed to have had three injections at the school; some were known to have had less than three injections, and in most the injections were missed entirely. Some parents and children confused diphtheria immunization with vaccination against smallpox and attempts at scarlet fever immunization. We realized the data were so unreliable that they could not be accepted for quotation. This statement is emphasized, for some belief seems to prevail that the value of diphtheria immunization is overestimated. This belief, based on what we regard as unreliable data, has been voiced by physicians of our own staff.

Summarizing the results of free school immunization we find that the diphtheria incidence decreased markedly when as many as 85 per cent of all school children had received immunization. This represents a large number, 36,718, but the 15 per cent not immunized represents in large part those unable to take care of themselves, for more children were brought to the free hospital. These children came principally from the homes of the poor, and very few cases of diphtheria occurred in the better residential districts of the city, even when the children attended private schools in which free immunization was not given.

Free school immunization to diphtheria as it has been practiced is for various reasons not the best solution of the diphtheria problem. Immunization in the schools makes people believe the protection is needed for the child at school, while we know that most of the diphtheria deaths occur in children before the school age. The well-to-do are better informed, are easily persuaded, and take advantage of the "free" part, not even having to give time to make a visit to the doctor. The poor require much more argument to convince them of its value, and it is not so pleasant for "workers" to convince them. While the parents may be willing to see to it that their children come for one injection, they will not make the effort two more times at one-week or three-week intervals. Furthermore, the poor move their homes frequently with the result that contact is lost and subsequent injections are not made.

Immunization of the younger children of the well-to-do is as essential as it is for the poor. Nearly 60 per cent as many adults with diphtheria were admitted to the hospital in the last five years as were children. These adults supply most of the less skilled types of labor in the markets, stores, and the homes where they expose the preschool children who, under a system of immunization at the school, have not yet been immunized.

CONCLUSION

It is unfortunate that with a method for immunization against diphtheria so effective, a plan for having it so far reaching as to be able practically to eliminate the disease has not been developed. School immunization to diphtheria does not solve the problem, nor does free immunization. Massive statistics are accumulated, and from large numbers false conceptions of thoroughness and security are developed. Lay organizations cannot solve diphtheria immunization. It is a medical problem to be solved by the doctor. What is needed is a method that gives a lasting immunity in a short time and requires preferably one injection. If such a method is not developed to get really good results diphtheria immunization will have to be compulsory at the age of six months or a year, and those not immunized by the physician will have to be taken care of by the Health Department. In any event the poor will have to be protected to get all the benefits the method can accomplish. The more fortunate financially will be protected, as they are educated more easily.

This study is presented with the hope it may be of value to pediatricians in other communities when pressure is brought for free school immunization by school boards, child welfare organizations and Parent-Teachers' Associations.

822 RIALTO BUILDING

ACUTE INFECTIOUS CROUP

A General Study of Acute Obstructive Infections of the Larynx, Trachea, and Bronchi With an Analysis of 727 Cases

A. Harry Neffson, M.D., and Samuel M. Wishik, M.D.
New York, N. Y.

PART III. CROUP ASSOCIATED WITH SPECIFIC DISEASES

The association of croup with acute infectious diseases is well known. It is impossible in the light of our present knowledge to state whether such croup is due to a specific infection by the same disease process which may be causing the rhinitis or pharyngitis or whether there occurs a secondary invasion by pyogenic organisms upon sites of lowered resistance. It has been deemed advisable by the authors to consider separately cases of croup intimately associated with specific infections.

CROUP WITH MEASLES

Slight hoarseness is a frequent occurrence in measles, but in the three-year period under study, out of 3,677 cases of measles, there were thirty-five patients, or slightly less than 1 per cent, who had enough laryngeal abnormality to give them symptoms of respiratory obstruction. These occurred between the months of February and July and between the ages of nine months and six years. There were twenty-one males and fourteen females.

Croup may occur at any stage of measles: during the incubation period (two cases); coincident with the acute symptoms of measles (twenty-eight cases); and during the convalescent period (five cases). At the time of exposure to measles and thereafter during the incubation period, children occasionally show malaise, slight temperature elevation, coryza, and cough. It is conceivable that such early catarrh can involve the larynx as well, even before the invasive period. Otherwise, the occurrence of croup at this stage must be considered merely a coincidence. One of these two cases was mild; the other, severe, requiring intubation and terminating in death.

Twenty-eight cases occurred during the eruptive stage. In ten the croup appeared at the same time as the rash; in two, before the rash (two and five days before). In two others, the croup occurred the day after the rash; in seven, on the third day; and in the rest, within six days after the eruption first appeared. These cases ranged through all degrees of severity.

Five cases occurred between ten and twenty-two days after the subsidence of the rash. These are considered part of the measles infection rather than pure coincidence because the measles cough and often bronchopneumonia as well still existed at the time the croup developed.

All three groups are considered together regardless of the time of occurrence in the measles cycle because of the general similarity in local pathologic findings and clinical course.

The development of marked respiratory difficulty is, as a rule, more gradual than in the usual nondiphtheritic infectious croup. It is often preceded by cough and hoarseness for from one to three days before the stridor sets in. The hoarseness is more severe than in the nonspecific type. Moreover, these cases show a greater degree of supraglottic edema in proportion to the other changes than in the other types. This is so common and conspicuous a feature that the disease has been called "acute laryngeal edema with measles" by some writers.[27] There is much congestion of all the laryngeal structures, and the subglottic swelling is a secondary feature compared to the prominent angry swelling of the aryepiglottic folds. Ulcerations of the larynx were found in four cases. The temperature corresponded to that of other types of croup except when it was elevated during the eruptive stage of the measles.

Direct laryngeal cultures were obtained in nineteen of the cases. Of these, fourteen showed *Staphylococcus aureus;* thirteen showed *Streptococcus hemolyticus;* and four, *Streptococcus viridans,* alone or in combination. The frequent occurrence of staphylococci is in sharp contrast to its occasional presence in the nonspecific croup series.

Anatomic Distribution.—There were twenty cases, or 57 per cent, in Group I; ten cases, or 29 per cent, in Group II; and five cases, or 14 per cent, in Group III.

All cases of measles have some degree of tracheitis and bronchitis, but in this grouping only those showing obstructing subglottic edema were put in Group II, and only those with definite bronchial obstruction into Group III.

Complications.—Order of Frequency:

Pneumonia	17 cases or 49 per cent
Myocarditis	7 cases
Otitis media	6 cases
Septicemia	3 cases
Bronchiectasis and lung abscess	1 case

Seventeen of the patients suffered from some complication or other.

Bronchopneumonia.—The bronchopneumonia was not different from that seen so often in the general run of measles cases. It was a constant feature in all but the mildest of these croups.

Anatomic Distribution.—In Group I four patients, or 20 per cent, had pneumonia; in Group II eight, or 80 per cent, had pneumonia; and in Group III five patients, or 100 per cent, had pneumonia.

Myocarditis occurred in seven patients, associated with pneumonia in all, with otitis media in two, and with septicemia in two. One patient had extreme supraglottic edema. Two patients received no mechanical intervention; two received suction; and three were given the combined suction-intubation treatment. All the patients died, and the clinical signs of myocardial damage were confirmed by postmortem examination in the three cases in which permission for necropsies was obtained.

Septicemia: Of the three cases, all associated with pneumonia, two died. A hemolytic streptococcus was isolated from the blood and larynx in two cases; *Streptococcus viridans*, from the other.

Bronchiectasis and Lung Abscess: The case report of the one patient with this complication is given.

A one-year-old baby boy was admitted with a history of pertussis for five weeks and a measles rash for one week followed by symptoms of croup. The child was restless and pale. There were slight to moderate retractions, slight stridor, moderate hoarseness, and extreme suppression of breath sounds, particularly over the right upper lobe. There was a fading measles rash. The temperature was 102.8° F.; pulse, 140; respirations, 54. Laryngoscopy showed great congestion and considerable edema of the supraglottic structures, which obscured the vocal cords and the subglottic region. A slight amount of mucopus was aspirated. Cultures from the larynx showed *Staphylococcus aureus* with *Streptococcus hemolyticus* and *viridans*. The next day both eardrums were incised and cultures of the pus showed *Staphylococcus aureus*. The following day, râles were heard at both bases, and breath sounds were diminished over the right lung. This and roentgen-ray examination led to the belief that there was a bilateral bronchopneumonia. During the next five days his chest condition seemed to clear up, and his breathing was unobstructed. On the sixth day he became almost aphonic and had loud stridor and deep retractions, greatly diminished breath sounds, and increased restlessness and pallor. Laryngoscopy showed a great amount of swelling and distortion of the epiglottis, aryepiglottic folds, and arytenoids and ulceration and swelling of the false cords. The vocal cords could not be seen. On aspiration a moderate amount of mucopus was removed, with considerable relief of obstructive symptoms. In the evening dyspnea became more severe; breath sounds, more distant; and restlessness increased. A laryngoscopy was performed, and the child coughed up a great deal of mucopurulent material. During the procedure he stopped breathing. He was intubated with a size nine-month tube, given artificial respiration, and revived. The next day the child suddenly stopped breathing and could not be brought back to life. Postmortem examination showed bronchopneumonia in the left lower lobe and the entire right lung. A bronchiectatic abscess was found in the right lower lobe; and diffuse bronchiectasis, in the right middle and right lower lobes. The epiglottis was eroded, and there was ulceration of the larynx with almost complete destruction of the vocal cords. Microscopic sections through the larynx showed an acute ulcerative process with adherent membrane of the stag-horn type suggesting a pseudodiphtheritic membrane. This, however, lacked the characteristics of diphtheria in that the basement membrane was destroyed, and the membrane was firmly attached to the underlying

structures. The inflammatory reaction, extending well out into the musculature, was characterized by a fibrinous precipitate, extreme edema, and considerable leucocytic infiltration.

TABLE III

TYPE OF TREATMENT REQUIRED

	GROUP I	GROUP II	GROUP III	TOTAL
Palliative Treatment	20	5	0	25
Suction	0	2	1	3
Suction-Intubation	0	3	4	7
Tracheotomy	0	0	0	0

The following case illustrates the use of aspiration in a case of measles with croup.

A one-year-old infant, sick for eight days with measles, was admitted to the hospital looking desperately ill, pale, cyanotic; he was breathing very rapidly, with deep suprasternal, substernal, and intercostal retractions. The voice was almost entirely gone. Breath sounds were diminished throughout, and there were scattered areas of dullness and râles in both lungs. The admission temperature was 105° F.; the pulse, 170; respirations, 72. Laryngoscopy showed thick mucopurulent exudate both above and below the cords. Quick suction of the larynx and trachea removed a large amount of thick, almost membranous exudate, and the infant was greatly relieved. His general condition, however, was so poor that he was not expected to live another twelve hours. He was given oxygen by nasal catheter, an alcohol sponge, and a low soapsuds enema. The next morning his condition was unbelievably improved. His temperature had fallen to 100.4° F.; he was breathing easily with hardly any retractions. During the next four days the bronchopneumonia became very extensive, involving both lungs. A blood culture at this time showed a *Streptococcus hemolyticus*. He was given frequent transfusions and kept in an oxygen tent. Both eardrums became infected, and a very intractable diarrhea developed. Nine weeks after admission the child's voice was still almost entirely gone. At this time laryngoscopy revealed more than moderate supraglottic edema obscuring the vocal cords. The child was discharged cured on the eighty-eighth day of illness. Seen about a year later, he was found to be in excellent health with a strong clear voice, except when crying when slight hoarseness was noted.

Suction alone is not often indicated in measles croups because exudation is not usually the most important factor in the obstruction. Although the exudation is copious, it does not often become dry or sticky enough to prevent its free elimination.

Intubation has been found almost uniformly unsuccessful in this series so far as life is concerned. Six of the seven patients intubated died. The immediate relief of obstruction by each intubation was satisfactory, but the subsequent course was always turbulent. Cough-up or blocking seemed a frequent occurrence. The patients were not as resistant to such episodes of anoxemia as they are with most other forms of croup. *All deaths were sudden and to a degree unexpected, usually not warranted by the apparent general condition of the patient and the degree of obstruction.* It is true that postmortem examinations

usually disclosed extensive infections of the lungs and often sepsis. Nevertheless, the authors have concluded after these experiences and in view of this analysis that tracheotomy should be considered early when the intubated measles patient suffers from obstructive asphyxial attacks.

TABLE IV

MORTALITY ACCORDING TO TREATMENT REQUIRED

	GROUP I	GROUP II	GROUP III	TOTAL	PERCENTAGE
Palliative Treatment	2	1	0	3	12
Suction	0	0	1	1	33
Suction-Intubation	0	2	4	6	86
Total Number of Deaths				10	29

Morbid Anatomy: Necropsies were performed on five of the ten patients who died. There were several outstanding features. In four cases there were extensive ulcerations of the larynx and trachea with exposure and erosion of the laryngeal cartilages and tracheal rings. In two of these the ulcerated areas were covered with pseudomembrane. Extensive bronchopneumonia was present in all five cases. In three cases, microscopic sections through the heart showed severe toxic granular degeneration of the myocardium. In two of these the liver, spleen, and kidneys were similarly involved. Cultures of the heart's blood showed *Streptococcus hemolyticus* in three cases, *Streptococcus viridans* in one case.

CROUP WITH SCARLET FEVER

Of 4,499 scarlet fever cases, nine developed croup, an incidence of 0.2 per cent. These occurred between the months of November and April in children ranging from two to twelve years of age. Six were males and three females.

In all but two cases the croup preceded the rash; in one patient, by six days; in two, by three days; in two others, by one day; and in the other two, by some hours. In the other two patients the croup developed five and fourteen days after the rash.

The disease resembles nonspecific infectious croup except for the fact that the temperature is usually higher. The average admission temperature was 103° F. Hoarseness is not an unduly prominent feature. The respiratory difficulty and cough may be the only symptoms at onset, or these may be associated with the prodromes of scarlet fever, such as headache, vomiting, and angina.

Bacteriology.—Cultures taken directly from the larynx showed a hemolytic streptococcus in five patients, *Staphylococcus aureus* in five, and a pneumococcus in one.

Anatomic Distribution.—In Group I there were two cases with no deaths; in Group II, four cases with one death (mongolian idiot); in

Group III, three cases with two deaths. The total number of deaths was three, a mortality of 33 per cent.

Even though the series is small, it would seem from the above that patients suffering from croup associated with scarlet fever are more severely afflicted than the average croup patients of the nonspecific infectious type.

Complications.—Otitis media occurred in four cases; pneumonia, septicemia, and myocarditis, in two cases each; and pneumothorax, mediastinitis, and submental abscess, in one case each.

Seven of the nine patients had some form of complication. The pneumothorax and submental abscess occurred in tracheotomized patients. A hemolytic streptococcus was the offending organism in both cases of septicemia.

TABLE V

TREATMENT REQUIRED

	GROUP I	GROUP II	GROUP III	TOTAL
Palliative Treatment	2	2	0	4
Suction	0	0	0	0
Suction-Intubation	0	2 (1 died)	1 (died)	3 (2 died)
Tracheotomy	0	0	2 (1 died)	2 (1 died)

The following case is typical.

A twelve-year-old boy came in on the third day of an illness starting with coryza and fever and followed by dyspnea, stridor, hoarseness, and cyanosis. On admission he was moderately hoarse, very dyspneic, breathing with loud stridor and moderate retractions. His color was good, but breath sounds were greatly suppressed throughout the chest. Laryngoscopy showed considerable supraglottic and subglottic edema and congestion. No membrane or exudate was seen. He received suction once, and, although little mucopus was obtained, he was greatly relieved. Breathing was easier, and the breath sounds were transmitted better. The temperature was 103° F.; pulse, 110; respirations, 30. Nine hours later he suddenly became extremely dyspneic, cyanotic, delirious; there were very deep retractions and practically no breath sounds. He was intubated with a size seven-year tube and was immediately relieved, going to sleep at once. At this time his temperature was 104° F. The next day there appeared a punctate erythematous rash; the appearance of the throat was suggestive of scarlet fever. He was given 10,000 units of scarlet fever antitoxin intramuscularly after negative sensitivity tests. The Schultz-Charlton reaction was positive later in the day. About ten hours after intubation, his tube became blocked with dried-up mucopus, and he was extubated with considerable relief. He did not require reintubation, but his general condition continued to be very poor; the heart sounds were very weak; and there was poor vasomotor tone. His temperature ranged between 105° F. and 106° F. He was given glucose intravenously, adrenalin and ephedrine subcutaneously, and was digitalized. His breathing continued to be unobstructed, but his heart became weaker; his temperature rose to 108° F.; and he expired on the third day after admission. Cultures from the larynx showed *Streptococcus hemolyticus.* Postmortem examination revealed recent verrucous vegetations on the mitral valve, congestion, and edema of the lungs with beginning broncho-

pneumonia in several places, suppurative mediastinal lymphadenitis, edema and congestion of the larynx, trachea, and bronchi, and acute splenitis. Cultures of the heart's blood showed *Streptococcus hemolyticus.*

CROUP WITH PERTUSSIS

Of 875 patients with pertussis, six developed croup, an incidence of 0.7 per cent. The ages of the patients were six, seven, and nine months and three, four, and five years.

In no case did croup occur before definite paroxysms of cough had set in. In one case croup began after a week of paroxysms; in another, after two weeks; in a third, after three weeks; in two others, after a month; and in the last case, four months after the onset of a paroxysmal cough which was still present at the time.

The admission temperature was 100° F. in three patients; 101° F., in two patients; and 103° F., in the other. The voice became hoarse; the cough, barking; and the coughing paroxysms, more difficult. Soon respiratory difficulty remained between, as well as during, the paroxysms. Subglottic edema was the chief offending factor in all cases, and, therefore, intubation, whenever it was resorted to, very effectively relieved the obstruction. Three of the six cases required such intervention. Although cough persisted while the tube was in situ, neither typical paroxysms nor whoop were heard after intubation. Likewise, cough-up of the tube was a less troublesome feature than would be expected—in fact, no more so than in croups not associated with pertussis. In two of the three cases intubated, the tubes were needed for relief of the obstruction for only a few hours. In the other case it was removed after nine days.

Bacteriology.—Cultures were obtained from the larynx in three cases showing *Streptococcus hemolyticus* in one, *Streptococcus hemolyticus* and *viridans* in a second, and *Streptococcus hemolyticus* and *viridans* and *Staphylococcus aureus* in the third.

Anatomic Distribution: In Group I there were three cases; in Group II, two cases; in Group III, one case.

Complications.—One patient had bronchopneumonia. The others were uncomplicated.

Prognosis.—All the patients recovered. The child who had been intubated over a period of nine days was seen six months after discharge and was found to be in good health with a normal voice.

CROUP WITH VARICELLA

Out of 1,616 varicella patients admitted to this hospital during the three-year period, only one, whose case is given below, developed respiratory obstruction.

A four-year-old boy was admitted on the second day of an illness which began with a varicella rash and was followed the same evening by progressively increasing

stridor. On admission the child looked acutely and desperately ill, was cyanotic, restless, and had very deep retractions above and below the sternum. There was a loud inspiratory stridor and a louder rattling expiratory stridor, which we have found characteristic in extreme supraglottic edema. This diagnosis was confirmed by laryngoscopy, which showed extreme swelling and distortion of the epiglottis and aryepiglottic folds, preventing visualization of the glottis. Ulcerations were seen on the epiglottis and the aryepiglottic folds. There were early varicella lesions all over the body. A tracheotomy was performed at once, and the patient was completely relieved. Cultures from the larynx showed *Streptococcus viridans*. During the next week the bronchi, repeatedly blocked with gummy mucopus, required frequent suction. The larynx did not return to its normal condition until sixteen days after admission. At that time the tracheotomy tube was removed, and the child was discharged cured five days later. Six months later when the child was seen, he had a strong, clear voice of normal pitch. There had been no recurrence of croup.

* * * * *

OTHER TYPES OF CROUP

A selected group of case studies which illustrate some of the less usual types of respiratory obstruction and indicate the diagnostic problems encountered are given below.

Laryngospasm in Tetany

A male infant, aged twelve weeks, was sent in with the diagnosis of possible laryngeal diphtheria. He had been ill with pertussis for the past seven weeks, but during the last week he had had no paroxysms. The day before admission he began to have spells of apnea with cyanosis. During the preceding two weeks, he had been gradually weaned from the breast and had been given one dram of cod liver oil daily. While being examined, he became extremely cyanotic and spastic, and he stopped breathing. After half a minute his color slowly returned, and breathing became easier. Carpopedal spasm was present. While the larynx was being studied through the laryngoscope, the child had another similar attack, offering the examiner an opportunity to witness the changes as they occurred. There was close approximation of the aryepiglottic folds and the false cords, obscuring the vocal cords and causing complete obstruction. Nearly a minute passed before the child began to breathe again, in short gasps spaced about five seconds apart. An estimation of the calcium and phosphorus in the blood showed a normal concentration of each. He was given hydrochloric acid milk, viosterol, and calcium. He had a few more of these spells during the next two days and then improved. He was discharged cured ten days after admission.

Bulbar Poliomyelitis

A seven-year-old girl was admitted on the fourth day of her illness, which began with a sore throat and vomiting followed by difficulty in swallowing, drooling from the mouth, nasal speech, and attacks of dyspnea and cyanosis. During the preceding two months she had been ill with pertussis. Two years before she had had an attack of anterior poliomyelitis, for which she had been treated at the Willard Parker hospital. She was discharged with a weakness of the left shoulder girdle. While she was being examined, she suddenly became very dyspneic and cyanotic; she had deep retractions, was extremely restless, and sat up on the table with arms outstretched. This spell lasted for about a minute; then breathing became easier. She had a nasal voice; being unable to swallow, she had a large accumulation of saliva

in the throat. There was no stiffness of the neck or back, and the Kernig, Brudzinski, and Babinski signs were all negative. There was no facial or palatal palsy. The only weakness present was that of the left upper extremity, which remained from her old attack of poliomyelitis. While a laryngoscopy was being performed, the child had two spells of severe respiratory distress during which the right aryepiglottic fold and right arytenoid were seen to be pulled into and closing the glottis. A spinal tap showed only thirty cells per cm. with 60 per cent polymorphonuclears and 40 per cent lymphocytes, a slight increase in the albumin and globulin, and a normal sugar content. The admission temperature was 102.4° F. This rose to 105.6° F. in twelve hours. The child died suddenly thirteen hours after admission. The death was not preceded by an attack of dyspnea. A blood culture on admission was negative. A culture from the larynx showed *Streptococcus viridans*. Postmortem examination showed evidence of an old and a new poliomyelitis infection of the pons, medulla, and spinal cord. The right recurrent laryngeal nerve showed beginning axonal degeneration and phagocytosis. The section included a ganglion, the cells of which were in various stages of degeneration and phagocytosis by microglia.

This case has been reported by T. B. Quigley[28] because of the second attack of poliomyelitis.

Papilloma of the Larynx

A two-year-old boy was sent in from another hospital with a diagnosis of diphtheritic croup. He had been given 20,000 units of diphtheria antitoxin. There was a history of progressive hoarseness and dyspnea for the past three days. On admission the child looked exhausted, pale and cyanotic; he was exceedingly dyspneic, aphonic, and had deep retractions. Breath sounds were entirely suppressed throughout. Laryngoscopy displayed a moderately congested larynx and what at first appeared to be membrane on the right vocal cord. After an unsuccessful attempt at removal by suction, a pair of alligator forceps was used. Only a few bits of rather friable tissue could be obtained. Arising just above the right vocal cord, there was a papillomatous growth which was drawn into the glottis with each inspiration and caused almost complete obstruction. Intubation was performed and gave immediate complete relief, the child falling asleep on the table. On being told that this was not a case of contagious disease, the parents asked to have the child transferred to another hospital where the growth was successfully removed.

Foreign Body—Jack in Esophagus

A four-year-old boy was sent in from another hospital with a diagnosis of croup. With him came a history of a cold and cough for two weeks and symptoms of croup two days before admission. On admission the child was very slightly hoarse and had no respiratory difficulty. Breath sounds were normal. A laryngoscopy showed slight supraglottic edema and congestion; the rest of the larynx was normal. There was no change in the child's condition until three days later when he refused food, developed a temperature of 104° F., and had rapid, somewhat labored respirations. A roentgen-ray picture taken at this time revealed the presence of an opaque foreign body in the neck. Fluoroscopy showed this to be a toy jack lodged in the upper part of the esophagus. On close questioning, the foreign-speaking parents said they thought that about three weeks before admission the child while playing had possibly swallowed a marble. At that time he was taken to a hospital where an attempt had been made to find it. However, nothing was found, and the parents were told to bring the child back in a week. On learning that the case was not

contagious, the parents withdrew the child from isolation. At another hospital, the jack was removed, but the patient died. The posterior wall of the esophagus had been eroded, and a mediastinal abscess had formed.

Retropharyngeal Abscess

A nine-month-old baby girl was sent in from another hospital with a diagnosis of diphtheritic croup and a history of fever for three weeks, stridor growing progressively worse for two days, and cough, dysphagia, dyspnea, and cyanosis. She had been given 10,000 units of diphtheria antitoxin. On admission the child looked acutely ill, pale, dehydrated, and undernourished. She was breathing rapidly; the head was thrown back; the voice was weak but clear; there was a loud respiratory stridor, but no retractions were seen. The breath sounds were obscured by the stridor. Digital palpation of the pharynx disclosed the suspected retropharyngeal abscess. This was incised, and an ounce of pure pus was obtained and removed by suction. The child was completely relieved. She was given a transfusion and a week later was discharged greatly improved.

Cervical Abscess and Pharyngeal Edema

A two-year-old girl, sick for two days, was sent in from another hospital with a diagnosis of diphtheritic croup. She had been given 20,000 units of diphtheria antitoxin. On admission she was acutely and desperately sick, very pale and cyanotic, extremely dyspneic with moderate retractions. Breath sounds were greatly diminished over the entire chest. The voice was weak but clear. The palate was tremendously swollen on the right side, but no membrane was present. Palpation revealed no evidence of a retropharyngeal abscess. On the right side of the neck there was a swelling from which pus was aspirated. An immediate tracheotomy was decided upon. As this was being performed, the child stopped breathing and turned ashy-white. The operation was stopped, and one of the operators performed a quick laryngoscopy and inserted an open metal suction tube into the trachea, creating an airway. Artificial respiration was given and the patient slowly revived. The tracheotomy was finished while the suction tube was kept in the trachea. The abscess of the neck was incised and drained, and several transfusions were given. For three days the child was in a desperate condition. The day after the tracheotomy there was a slight degree of pneumothorax on the right side. This cleared up in a few days. The tracheotomy tube was removed eight days after the operation, and the child was discharged cured five weeks later.

Lymphosarcoma and Acute Lymphatic Leucemia

A four-year-old negro was sent in from another hospital with a diagnosis of diphtheritic croup. The preceding ten days the child had had a running nose and a cough. A few days later he became short of breath; this condition continued to grow worse up to the time of admission. The past history was entirely negative. On admission the child's breathing was rather rapid and labored with prolonged expiration, but no retractions were seen. The voice was clear. A circular mass 4 inches in diameter was noted to the left of the manubrium sterni. It was soft, fleshy, not fluctuant and prevented palpation of the underlying ribs. Over the left upper lobe anteriorly the percussion note and breath sounds were diminished, and bronchial breathing was present. The left tonsil looked ulcerated; the right tonsil was peculiarly pale and shiny, and blood vessels could be seen running across the upper pole. The edge of the spleen was felt almost down to the brim of the pelvis; and the liver, down to the umbilicus. The cervical, axillary, and inguinal glands were hypertrophied. A diagnosis of lymphosarcoma or lymphatic leucemia was

made. The latter diagnosis was confirmed by the blood examination which showed the following: hemoglobin, 50 per cent; R.B.C., 3,730,000; W.B.C., 452,000; polymorphonuclears, 2 per cent; large lymphocytes, 62 per cent; small lymphocytes, 8 per cent; lymphoblasts, 28 per cent. A rare myelocyte was seen. The platelet count was 62,800. The Wassermann test was negative. Roentgen-ray examination showed a sharply defined opacity obscuring the central shadow and almost the entire left lung field, filling in the retrosternal space, and sheathing the heart and great vessels. The child became progressively weaker and more dyspneic; the lymph glands, liver, and spleen became larger; the child died seventeen days after admission. Postmortem examination showed a large mediastinal mass of infiltrating, neoplastic tissue 15 cm. by 18 cm. On section it was found to contain many lobulated areas suggesting lymph nodes, embedded in greyish yellow, elastic tumor tissue, extending through the chest wall and involving the first four interspaces and infiltrating the intercostal and pectoral muscles. There was leucemic infiltration of the lungs, liver, spleen, kidneys, muscles, and epicardium.

Foreign Body in Trachea

An eight-year-old boy was admitted with a history of severe coughing, which had begun about ten days before admission and had been followed by paroxysms of coughing and emesis. A tentative diagnosis of pertussis was made, and the child was admitted for observation. During the next two weeks after admission, he had occasional coughing spells and vomiting, but no "whoop." The blood count was not typical of pertussis. A roentgenogram was normal. On the twenty-eighth day of his illness, he became somewhat croupy. The next day one of the authors was called up to see the child in a hurry. He was found to be hoarse, with a croupy cough, very dyspneic, with deep retractions and cyanosis. Breath sounds were absent throughout. Laryngoscopy showed a normal larynx, but just below the cords the edge of a foreign body was seen. This was removed with an alligator forceps and found to be a pistachio nut. The breathing became normal at once. The boy said he had eaten these nuts at the time he first started coughing. No pulmonary complications ensued.

Mucous Plugs in the Larynx and Trachea in Pertussis

A four-year-old girl was sent in with a diagnosis of croup and a history of a "cold" for one week, spasmodic coughing for three days, and severe dyspnea, orthopnea, and cyanosis for the preceding twenty-four hours. On admission the child was greatly distressed, sitting up, breathing with great difficulty but without retractions, and looking mildly cyanotic. The voice was clear. There was a peculiar, high-pitched cough simulating a sneeze but different from her usual sneeze. The facies suggested pertussis, the eyes being watery and red. The throat was mildly congested and full of thick mucoid secretions. The chest was full of coarse, bubbling râles. Laryngoscopy revealed the larynx to be normal except for the presence of thick mucoid secretions. Aspiration of the larynx and trachea removed a large amount of the mucus and gave considerable relief. On auscultation of the chest, râles could no longer be heard. During her stay at the hospital, she had several attacks of severe dyspnea during which her temperature would go up, once as high as 105.8° F. Following each laryngoscopy and suction, she would be greatly relieved, and the temperature would drop precipitously. Roentgenograms of the chest suggested bilateral bronchiectasis. After continued questioning, the mother finally told us that one of her other children had been coughing, whooping, and vomiting for about a month, but her family doctor had told her that the child had only a "cold." After five weeks the child was discharged greatly improved.

Dried Blood Clots in the Larynx and Trachea

A fifty-three-year-old man was sent in from another hospital with the diagnosis of diphtheritic croup. There was a history of hoarseness for three weeks and progressively increasing dyspnea for ten days. On admission he was completely aphonic, extremely dyspneic, orthopneic, and moderately cyanotic. There were deep suprasternal and supraclavicular retractions on inspiration with strong overaction of the accessory muscles of respiration. Breath sounds were diminished throughout, particularly over the upper lobes. The heart sounds were of poor quality, and the first sound at the apex was split. There was a marked "aspiration" pulse. The lower extremities were moderately edematous. The tongue was dry, red, leathery. The entire palate was covered with a dirty, sticky, sanguinous, mucopurulent exudate which extended onto the tonsillar pillars, uvula, and pharynx. An indirect laryngoscopy was unsatisfactory, the entire region being covered with this exudate. Direct laryngoscopy showed this material to extend down below the vocal cords, practically filling the entire lumen. Suction was applied, and many pieces of exceedingly sticky, bloody material were removed. This gave the patient a good deal of relief. He was put to bed; fluids were pushed; syrup of ipecac was given; oxygen was administered; and rapid digitalization was begun. After an hour, he fell asleep breathing with hardly any effort, his color and pulse greatly improved. Roentgen-ray examination showed an enlarged heart and diminished aeration of the left lung. The blood count was: R.B.C., 2,600,000; hemoglobin, 65 per cent; W.B.C., 16,500 with 88 per cent polymorphonuclears and 12 per cent lymphocytes. The blood sugar was 125 mg.; the N.P.N. 153, mg. per 100 c.c. The urine contained large numbers of red cells and showed three-plus albumin; otherwise it was normal. The blood pressure was 175 during systole and 90 during diastole. There was no renal tenderness.

That evening his heart became quite irregular with many dropped beats. The next morning he coughed up a black mass looking very much like a prune. His breathing was effortless at this time, and breath sounds were well transmitted throughout. His heart was regular, and its quality was improved. That afternoon he suddenly took a turn for the worse. Auricular fibrillation had evidently set in, and the urine was full of blood. His condition continued to get worse; he became pulseless; his heart sounds were inaudible, and he died of heart failure the next morning. His temperature ranged between 99° F. and 100° F. until eight hours before he died when it dropped to 96° F. A culture from the larynx showed a hemolytic streptococcus. A postmortem bronchoscopy showed that the larynx, trachea, and bronchi were perfectly clear with hardly any evidence of inflammation. Unfortunately, permission for a necropsy could not be obtained, and the cause of the bleeding could not be determined.

Fungus Infection of Larynx, Trachea, and Bronchi

A white baby boy, aged twenty-two months, was admitted to the hospital on the fifteenth day of illness. There was a history of fever and occasional cough until the day before admission, when he became croupy. On admission the child looked desperately sick, was pale and moderately cyanotic, and had mild substernal retractions. The voice was clear. He was underdeveloped and undernourished. Loose, white, patchy material covered the buccal mucosa, the undersurface of the tongue, the soft palate, and the pharynx. On laryngoscopy the same material was seen above and below the vocal cords and was easily removed by suction. This gave the child considerable relief. Examination showed scattered areas of dullness and râles throughout both lungs. Roentgen-ray examination at this time confirmed our diagnosis of bilateral bronchopneumonia. Bacteriologic examination of

the material in the mouth and larynx showed *Monilia albicans*. The child required a tracheal suction four days and again eight days after admission. On both occasions a good deal of the peculiar exudate was removed. He developed bilateral otitis media four days after admission and signs of meningeal irritation on the next day. Bacteriologic examination of the pus from the ears and the spinal fluid showed the presence of *Monilia albicans*. A blood culture taken two days after admission showed hemolytic streptococci. The child died twelve days after entry to the hospital. Postmortem examination showed bilateral bronchopneumonia; the bronchioles and alveoli were filled with exudate containing Monilia organisms. The brain was soft, edematous and congested, and covered in places with yellowish-white, gelatinous exudate. Cultures showed the presence of Monilia organisms. For a complete report of this case, see the paper by L. W. Smith and M. E. Sano.[29]

Vincent's Infection of the Mouth and Throat With Laryngitis

A two-year-old boy was sent in from another hospital with a diagnosis of diphtheritic croup. He had been given 20,000 units of diphtheria antitoxin. There was a history of a head cold for two weeks, noisy breathing for eight days, hoarseness for seven days, and dyspnea for three days. On admission he was slightly hoarse and dyspneic with mild substernal retractions and loud stridor. There were ulcerations on the lips, buccal mucosa, faucial pillars, palate, and tonsils. No laryngoscopy was performed on admission. Smears from the mouth and tonsils showed many fusiform bacilli and a few spirillae. The next day the lesions in the mouth and throat were worse, and the dyspnea was more severe. Laryngoscopy revealed a moderate degree of congestion and edema above and below the vocal cords but no ulceration. Smears were taken; the larynx was swabbed with a 33 per cent solution of sulpharsphenamine; and 0.2 gm. of sulpharsphenamine was given intramuscularly. A mouth wash of sodium perborate, peroxide, and sodium bicarbonate was ordered. Cultures from the larynx showed *Staphylococcus aureus* and rare *Streptococcus hemolyticus*. The smear did not show any fusiform bacilli or spirillae. Two days later the intramuscular injection of 0.2 gm. of sulpharsphenamine was repeated. The next day when the dyspnea increased and the retractions were deeper, it was feared a tracheotomy might be necessary. Laryngoscopy revealed an increase in the supraglottic edema and slight ulceration of the aryepiglottic folds and false cords. The larynx was again swabbed with a 33 per cent solution of sulpharsphenamine. The next day his condition remained about the same, and the intramuscular injection was repeated. The lesions in the mouth and tonsils and the breathing improved gradually; the child was discharged cured on the sixteenth day after admission. Even though smears from the larynx did not show the presence of Vincent's organisms, we feel that the laryngeal infection was an extension of that in the mouth and tonsils, because the course of the disease was entirely different both in its onset and decline from that of acute nonspecific infectious croup and because the severity of the mouth and laryngeal symptoms ran a parallel course.

Abscess of Larynx With Erysipelas

Three weeks before admission, a nine-month-old male infant was in another hospital because of erysipelas of the face complicated by bilateral acute purulent otitis media. When the erysipelas subsided a week later, the child became croupy. Nevertheless, a few days afterward he was taken home where he grew progressively more dyspneic. He was brought to a second hospital where he was observed for a day, was examined by laryngoscopy, and was given diphtheria antitoxin; he was sent to us with a diagnosis of laryngeal diphtheria. On admission the infant was desperately sick, cyanotic, with labored respirations, moderate retractions, and

greatly suppressed breath sounds, particularly over the left lung. There were draining and fluctuating abscesses of the scalp, the chest wall, the back of the neck, and the thighs. Numerous pustules and urticarial lesions were scattered all over the body. The voice was only moderately hoarse. Digital palpation disclosed a large swelling in front and to the left of the epiglottis, distorting the entire topography. Laryngoscopy showed extreme edema and distortion of all the laryngeal structures to the extent that the glottis could barely be seen. A diagnosis of abscess of the larynx was made, and tracheotomy was performed. This gave complete relief. Following that, the abscess was incised through the laryngoscope, and more than an ounce of pus was released. Cultures from the larynx, the blood, and skin abscesses showed hemolytic streptococci. The bronchi became blocked repeatedly with gummy mucopus requiring frequent removal. Three transfusions were given, but the child's condition became progressively worse, and he died on the fourth day after admission. On necropsy evidences of generalized sepsis in all organs were found.

Agranulocytosis

A twenty-eight-year-old woman was sent in to us from another hospital with a diagnosis of diphtheritic croup. She had been perfectly well until one week before admission when she awoke with "a sore throat and felt dizzy." Two days later feeling worse, she called a physician, who said that she had tonsillitis. Then her condition improved somewhat until the night before admission, when she became weak, aphonic, feverish; she complained of pain in her right chest on breathing and could not swallow. Physical examination showed an underdeveloped, undernourished white woman acutely and desperately ill, toxic and septic, with flushed face, dilating alae nasi, strongly acting accessory muscles of respiration, but no retractions. The voice was completely gone. The right tonsil was covered with thick, dirty white, tenacious membrane which extended onto the palate and the lower molar region. Its removal caused no bleeding. The heart sounds were very rapid and of poor quality; the percussion note and breath sounds were diminished over the middle of the right chest, but no râles were heard. She was extremely weak. Her temperature was 106° F.; pulse, 170; respirations, 60. A tentative diagnosis of agranulocytic angina was made, and an emergency blood count taken. This confirmed the diagnosis and showed the following: hemoglobin, 55 per cent; R.B.C., 3,800,000; W.B.C., 350; platelets, 320,000; polymorphonuclears, staff, 6 per cent; polymorphonuclears, segmented, 0 per cent; lymphocytes, 75 per cent; monocytes, 3 per cent; myelocytes, neutrophilic, 7 per cent; myeloblasts, 3 per cent; plasma cells, 3 per cent; histiocytes, 3 per cent. She failed to rally after transfusion and died shortly after admission.

A postmortem laryngoscopy showed ulcerative membrane on the right aryepiglottic fold, right arytenoid cartilage, and right vocal cord and ulceration of the left vocal cord and subglottic region. Necropsy revealed the following:

1. Agranulocytic angina involving the regions described above. In addition there was ulceration of the upper trachea and ropy plugs of mucopus in the secondary bronchi.

2. Lobar pneumonia of the right middle and the upper part of the right lower lobe.

3. Interlobar plastic pleurisy on the right side.

4. Hemopericardium with petechiae. A blood culture during life showed a slowly growing *Streptococcus viridans*. The throat culture showed *Streptococcus viridans* and *Staphylococcus albus*.

DIFFERENTIAL DIAGNOSIS

It is amazing how many different types of conditions are sent in to this institution wrongly diagnosed as croup not only by the private general practitioner and pediatrician, but also by the staff members of medical, pediatric, and otolaryngologic departments of leading institutions in this city. Many conditions which caused any type of respiratory difficulty or irregularity have been so misdiagnosed.

The following classified list gives the cases sent into the hospital as croup, usually as diphtheritic croup:

I. Throat infection
   - A. Nasopharyngotonsillitis and uvulitis
   - B. Peritonsillar abscess
   - C. Retropharyngeal abscess

II. Neurogenic disturbances in respiration
   - A. Poliomyelitis (intercostal, diaphragmatic involvement)
   - B. Convulsions (tetany, epilepsy, meningitis, pertussis)
   - C. Coma (diabetes, etc.)

III. Thoracic conditions altering respiration
   - A. Pneumonia
   - B. Pertussis
   - C. Asthma
   - D. Cardiac disease

SUMMARY AND CONCLUSIONS (PARTS I, II, AND III)

1. A plea is made for a standardized terminology of croup, and an etiologic classification is given.

2. Out of 727 consecutive cases of acute infectious croup treated at the Willard Parker Hospital between June 1, 1931, and June 1, 1934, 36 per cent were due to diphtheria; 55 per cent, to nonspecific infections; 5 per cent were associated with measles; 1 per cent, with scarlet fever. There has been a yearly rise in the proportion of nondiphtheritic croup cases admitted to the hospital. Nevertheless, most patients are still sent into the hospital diagnosed as diphtheritic croup.

3. The age of diphtheritic croup patients ranged from five months to fifty-three years; 22 per cent of the cases occurred in the second year of life; 77 per cent, under five years. In the nonspecific cases, the ages ranged from three months to fifty years; 46 per cent were in the second and third years of life; 78 per cent were under five years.

4. The local obstructive factors in diphtheritic and nonspecific croup are the same: namely, membrane formation, mucopurulent exudation, edema, and spasm. They vary only in degree and frequency of occurrence in each type.

5. The chronology of symptoms in diphtheritic croup is cough, hoarseness, and dyspnea; in nonspecific croup it is cough, dyspnea,

and hoarseness. In diphtheria there may be considerable hoarseness with only slight dyspnea, whereas in the nonspecific croup the dyspnea is usually out of proportion to the mild or moderate hoarseness.

6. A single, direct laryngeal culture involved an error of at least 21 per cent in the diagnosis of diphtheria; by the use of two laboratories it was reduced to 11 per cent. Reliance for diagnosis upon a single, though direct, culture from the larynx is therefore dangerous. In the nonspecific croups some type of streptococcus was found alone or in combination with other organisms in 92 per cent of the cases reported. A hemolytic streptococcus was the most frequent organism. It was present alone or in combination in 70 per cent, in pure culture in 28 per cent of the cases reported.

7. The complications of diphtheritic and nonspecific croup are classified and discussed.

8. A detailed account of the medical and operative treatment of both types of acute infectious croup is given with the rationale, the indications and contraindications, the technic, and the dangers. Cases illustrating the different points and methods of treatment are frequently interpolated.

a. Diagnostic laryngoscopy is indispensable, and diagnostic suction is often invaluable in the treatment of croup.

b. The suction treatment has eliminated the need for intubation in many cases and is an essential adjunct when intubation is required. It has removed the danger of sudden asphyxia which formerly attended intubation because of the jamming down of membrane. The authors feel that by its use both morbidity and mortality have been materially reduced.

c. Bronchial suction can be performed quickly and harmlessly without the use of a bronchoscope.

d. Repeated suctions and intubations properly performed are not harmful in either type of croup. In diphtheritic croup, 46 per cent required suction; 16 per cent, suction-intubation. In nonspecific croup, 7.3 per cent required suction; 5.3 per cent suction-intubation. The average number of suctions in nonintubated diphtheritic croup cases was 2.1; in intubated cases, 4.3; in nonintubated nonspecific cases, 1.2. The average number of intubations in diphtheritic croup was 2.9; in nonspecific croup, 3.2. In either type not more than 16 suctions and 12 intubations were required under any circumstances.

e. The intubation tube may be allowed to remain in the larynx as long as three weeks without danger of permanent damage. In diphtheritic croup the intubation tube remained in situ from nine hours to nineteen days, or an average of six days.

In nonspecific croup, the tube was in the larynx from three hours to fifteen days, the average being 5.3 days.

f. When intubation, with or without suction, effectively relieves the obstruction, it is to be preferred to tracheotomy in either type of croup.

g. The combined suction-intubation treatment of diphtheritic croup has eliminated practically entirely the need for emergency tracheotomy. When a tracheotomy is performed, it is done by choice because of too frequent cough-up or block-up of the tube followed by asphyxia, or when there is danger of the child becoming a chronic tube carrier. Only four tracheotomies were performed in this series. These were performed because the tubes did not give enough relief. All died, and the necropsies performed in three cases showed membrane extending down to the bronchioles and even lining the alveoli.

h. In nonspecific croup cases, eight tracheotomies were performed by choice—five because of block-up, three for cough-up, and none for chronic tube danger. The tracheotomy tube was removed after an average period of fifty days in the three patients who recovered.

i. Emergency tracheotomy in nondiphtheritic laryngitis, laryngotracheitis, and laryngotracheobronchitis is indicated only when there is extreme supraglottic edema which would overlap the intubation tube and render it useless. In ten cases this was necessary. The tracheotomy tube was successfully removed after an average period of sixteen days in the five patients who recovered. Extreme supraglottic edema can be diagnosed by the unique breathing, characterized by a low-pitched inspiratory stridor followed by a louder low-pitched expiratory rattle similar to a snore. The diagnosis can be confirmed by digital examination, without the need for laryngoscopy.

j. The frequency of the various methods of treatment required and the results obtained with them in the different types of croup are tabulated and discussed.

9. The important prognostic factors in diphtheritic croup are age, temperature elevation, extent of the infection and toxemia. In nonspecific croup, the single important factor is the extent of the infection.

10. The mortality in diphtheritic croup was 12 per cent; in the toxemic cases it was 64 per cent; in the nontoxemic cases, 6.8 per cent. The mortality in nonspecific croup was 4.8 per cent; in the laryngotracheobronchial group, 68 per cent. The deaths are discussed from a clinical angle and from postmortem findings.

11. Thirty-eight patients with the most severe cases of both diphtheritic and nondiphtheritic croup were seen from three months to a

year after discharge. There was a history of recurrent attacks of mild croup in six patients; five were slightly hoarse when crying; and five had a slight lowering of vocal pitch.

12. Out of 3,677 cases of measles, thirty-five developed respiratory obstruction. These are discussed in some detail. The mortality was 29 per cent.

13. Out of 4,499 cases of scarlet fever, nine developed croup. These are discussed in the same detail. The mortality was 33 per cent.

14. Out of 875 cases of pertussis, six developed croup. Three of these required intubation. There were no deaths.

15. Out of 1,616 cases of varicella, one case was complicated by croup. This is described.

16. There are cited fourteen interesting cases to illustrate diagnostic problems encountered. These include the following: tetany with laryngospasm; acute anterior bulbar poliomyelitis; papilloma of the larynx; foreign body in the esophagus; retropharyngeal abscess; cervical abscess and cellulitis of the pharynx; acute lymphatic leucemia; agranulocytosis; foreign body in the trachea; mucous plugs in the larynx and trachea in an unusual case of pertussis; dried blood clots in the larynx and trachea; fungus infection of the larynx, trachea, and bronchi; Vincent's infection of the larynx; and abscess of the larynx in a case of erysipelas and septicemia.

17. Diseases which are often mistaken for croup are tabulated.

18. As a result of our following the indications and methods given in this paper, the need for mechanical intervention in general and tracheotomy in particular has been lessened and the morbidity and mortality have been materially lowered. However, the method of treatment advocated here is practicable only in hospitals where proper facilities and expert operators are available *at all times*.

REFERENCES (PART III)

27. Oliver, K. S., and Turner, E. L.: J. A. M. A. 101: 1801, 1933.
28. Quigley, T. B.: J. A. M. A. 102: 752, 1934.
29. Smith, L. W., and Sano, M. E.: J. Infect. Dis. 53: 187, 1933.

Through error, the paper of H. R. Litchfield and R. P. Hardman, "Suction in the Treatment of Laryngeal Diphtheria," J. A. M. A. 80: 524, 1923, was omitted from Part I. This work at the Willard Parker Hospital preceded that of Gover and Hardman.[14]

FOOT OF EAST FIFTEENTH STREET, NEW YORK CITY
FLUSHING, NEW YORK

IDIOSYNCRASY TO AMMONIATED MERCURY OINTMENT

Report of Two Cases

Paul Harper, M.D.
New Haven, Conn.

THE use of ammoniated mercury ointment is so common that it seems worth while to emphasize the danger of idiosyncrasy.* In the two cases here presented the severity of the reaction as well as the peculiar swelling and cyanosis of the hands and feet is distinctive. These two patients are of added interest since the literature of the past one hundred years describes only five cases of poisoning* from local application of the drug. During the same period there are reports of fourteen cases of poisoning from ammoniated mercury taken by mouth.[4, 6, 7, 9, 10, 12, 14, 18, 19, 21, 22, 24] The symptoms were those of acute gastroenteritis and collapse with stomatitis in some of the less acute cases. There were four deaths. It is probable that a toxic reaction from local application is more common than the literature would indicate, especially as we have recently learned of four cases[16] in addition to those reported here—one generalized reaction and three cases where the reaction was localized to the area of application. In Table I are summarized data from the previously reported cases and from the two following records.

Case 1.—M. A. (Hosp. Hist. 44368), white female, aged twenty months. The family and past history were noncontributory. The present illness began May 8, 1926, with the appearance of a few vesicles on the feet. Ichthyol ointment was applied for several days; the eruption spread slowly. On the eleventh day, the patient was seen in the New Haven Hospital Dispensary and presented excoriated impetiginous lesions over the dorsa of both feet and a similar lesion behind the left ear, also a few vesicles on the legs and thighs. Ammoniated mercury, 10 per cent, was applied. That evening a generalized rash appeared. The next morning, May 19, the child was admitted to the hospital. Temperature, 38.2° C. The child was well developed and nourished. In addition to the impetiginous lesions already described she presented a papulovesicular eruption on a thickened erythematous base over the entire body except the scalp. There were small excoriated areas over the chest and neck exuding serum. The toes and the dorsa of both feet were red, swollen, and almost denuded of epithelium, leaving a raw, weeping surface. There was a crusted, moist exudate about the left ear. The lips were fissured, the buccal mem-

From the Department of Pediatrics, Yale University School of Medicine and the Pediatric Department of the New Haven Hospital.

*"Idiosyncrasy" is the term used in writing of the two cases here reported for reasons given in the section entitled "Comment." "Poisoning" was the term used in the majority of cases from the literature and will be used in this paper when referring to those cases.

branes were reddened and there was an erythematous eruption on the hard and soft palate. There was slight general glandular enlargement. The liver was down two fingerbreadths; the spleen was felt at the left costal margin.

Laboratory Data.—Blood Wassermann and tuberculin (1/10 mg.) tests were negative. Red blood cells, 5,000,000 per c.mm.; hemoglobin, 80 per cent (Sahli); white blood cells, 15,000 per c.mm.; polymorphonuclears, 58 per cent; lymphocytes, 27 per cent; large mononuclears, 6 per cent; eosinophils, 9 per cent. Repeated urine examinations were negative.

Treatment and Course.—The patient was restrained to prevent scratching. Boric acid compresses were applied to the excoriated surfaces, vaseline to the rest of the body. Improvement was rapid and the skin was nearly normal by the twenty-fourth day, June 1.

Diagnostic Skin Tests.—On June 1 a small amount of ammoniated mercury was applied to an area 5 cm. in diameter below the right scapula. Within six hours there was an erythematous blush over the back; within twelve hours this had become a minute vesicular eruption over the back and neck; and after twenty-four hours there was a papulovesicular eruption over the entire torso which gradually cleared during the next few days. Similar tests with ichthyol ointment and with vaseline gave no reaction. The patient was discharged cured on the thirty-sixth day. In spite of warnings the child received small applications of ammoniated mercury on two subsequent occasions with resultant reactions which were less marked but similar to that described above.

CASE 2.—P. M. (Hosp. Hist. A33207), white male, aged sixteen months. The family and past history were irrelevant. The present illness began Feb. 9, 1934, with a single impetiginous bleb on the chin. New lesions continued to appear on the chin and around the nose. Ammoniated mercury, 10 per cent, was applied to the lesions on the fourth day and was reapplied several times daily for nine days. Each new application was added to the previous layer which was not wiped off except as the child removed it by rubbing. On the ninth day a red macular rash appeared over the body, and the face became swollen. A tentative diagnosis of German measles was made. On the tenth day the child became worse; temperature, 38.3° C.; hand, swollen and cyanotic; increasing swelling of the face extending to the neck. On the twelfth day: swelling and cyanosis of the feet, otherwise unchanged. Ammoniated mercury discontinued. There was little change during the next two days, and the child was admitted to the New Haven Hospital on the fifteenth day, February 23. Temperature, 38.6° C. The child was acutely and seriously ill. There was a raised morbilliform rash on a thickened erythematous base over the whole body. The face was so swollen that the eyes were seen only through the slits between protuberant eyelids. The lips also were puffy. There was a generalized desquamation, and many crusted fissures were oozing serous fluid which was sanguineous in certain areas. There was marked enlargement of the cervical glands but no tenderness. The teeth were dirty, and the gums were swollen, dusky, and bled easily. The mucous membranes were reddened and showed small ulcerated areas on the palate, pharynx, and tongue. The hands and feet were so greatly swollen as to suggest danger of bursting the tightly stretched skin, which varied in color from blue to nearly black. The skin was peeling in sheets from both hands and feet, leaving a raw, weeping surface. There was moderate general glandular enlargement but no photophobia, no lacrimation, no hypotonia, no muscle weakness. The spleen was felt at the costal margin. The liver was down 1 cm.

Fig. 1 is a photograph taken on the third day in the hospital. The shiny appearance of the hands gives some indication of the tenseness of the swelling. Desquamation of the fingers may be seen.

TABLE I

YEAR	AUTHOR	AGE IN YEARS	AMMONIATED MERCURY			TOXIC MANIFESTATIONS					RESULT	REMARKS
			PER CENT	AREA COVERED	LENGTH OF TIME USED	RASH	DESQUAMATION	STOMATITIS	EDEMA	RENAL LESION		
1883	Deakin[5]	50	7½	Feet	48 hr.	Generalized				0	Cured	Chronic eczema of feet became acute.
1883	Idem	55	7½	Feet	48 hr.	Face, scalp			Face	0	Cured	Chronic eczema of feet became acute.
1884	Green[3]	Adult	10	Scalp	24 hr.		Scalp, hands	----	Face, hands	0	Cured	Blisters between fingers.
1921	Bechet[2]	Adult	2½	Psoriasis lesions over body	14 days			----		No note	Cured	Not observed during acute stage.
1923	Alexander and Mendel[1]	40	5	Face, neck, arms	6 yr.			----		0	Cured	Other symptoms: fever, diarrhea, weakness, alopecia.
Present report		15/6	10	Legs, feet, ear	24 hr.	Generalized	Face, hands, feet	----	Face, hands, feet	0	Cured	Cyanosis of hands and feet.
Present report		11/3	10	Face	9 days	Generalized	Generalized	----	Face, hands, feet	0	Cured	Blue color of hands and feet.

Laboratory Data.—Kahn and tuberculin tests were negative. Red blood cells, 5,000,000; hemoglobin, 85 per cent (Sahli); white blood cells, 14,000 per c.mm.; polymorphonuclears, 52 per cent; lymphocytes, 36 per cent; large mononuclears, 6 per cent; eosinophils, 1 per cent; unclassified forms, 5 per cent. Repeated urine examinations were negative. Nonprotein nitrogen, 33 mg. per cent; blood pressure, 100/65.

Treatment and Course.—The child was placed without clothes under a light cradle which was maintained at a temperature of 26.6° C. He was restrained to prevent scratching. A thin coating of sweet oil was applied twice daily. The child was afebrile after the third day. Desquamation proceeded, and the skin gradually cleared. The swelling of face and extremities slowly subsided, as did the cyanosis of the latter. The fissures healed; the mouth improved; and the ulcerated areas cleared. Itching, which was not present on admission, became an increasing feature during the early convalescence and then subsided.

Diagnostic Skin Tests.—On the twenty-fourth day, March 4, 2 per cent ammoniated mercury was applied to an area on the chest 5 cm. in diameter. After fifteen hours there was a raised, rough erythematous eruption over this area. At this time 5

Fig. 1.

per cent ammoniated mercury was applied to a similar area on the opposite side of the chest. After six hours this region also showed a raised, rough erythematous surface. Within twelve hours there was a pronounced erythema over the chest and abdomen and at the end of eighteen hours, there was slight swelling and blueness of the right foot and questionable swelling of the right hand. The itching recurred to a marked degree, and the test areas desquamated a second time. Control tests with vaseline, lanolin, and mineral oil were negative. The same ammoniated mercury applied to another baby gave no reaction. The patient was discharged cured on the thirtieth day, March 10.

DIAGNOSIS

In the differential diagnosis acrodynia, arsenic poisoning, pellagra, and septicemia with metastatic localization were also considered. Acrodynia was suggested by the bluish red swelling of the hands and feet, the desquamation, the generalized rash, the stomatitis, and the irritability. Against this diagnosis were the relatively rapid onset, the short course, the absence of hypotonia, photophobia, sweating, and anorexia. More-

over, the normal nonprotein nitrogen and blood pressure are unusual in acrodynia.

Arsenic poisoning as seen in the epidemic amongst the beer drinkers of Manchester, England,[15, 17, 23] showed a combination of skin rash, stomatitis, facial puffiness, peripheral neuritis, cyanosis and edema of the hands and feet, which early in the epidemic was reported as erythromelalgia[17] and beriberi.[16] The lack of known exposure to arsenic, the absence of peripheral neuritis, the brief course, and the skin tests were all in opposition to this diagnosis in our patients.

Pellagra could account for the dermatitis, stomatitis, and the swelling of the extremities, but probably not for the blueness of the hands and feet. Both children had had adequate diets. Pellagra is rare in children under two years of age and was rendered unlikely in these cases by the lack of central nervous system symptoms and by the rapid improvement following the withdrawal of ammoniated mercury without the use of a diet rich in vitamin G.

Septicemia might have caused the fever, the rash, and the desquamation. The swelling of the hands might have been due to metastatic foci of infection. This diagnosis was not long considered in view of the stomatitis, the low fever, the absence of leucocytosis, the negative blood cultures and the course of the disease.

The diagnosis of ammoniated mercury poisoning depended upon the history of the use of the drug, the recognition that such a reaction was possible, the confirmatory evidence of the skin tests, and, in one case, the recurrence of symptoms following subsequent application of ammoniated mercury. Further confirmation by analysis of the feces and urine for mercury would have been desirable. However, it seemed that the diagnosis was already clear. Skin tests with a more easily absorbed form of mercury were suggested but did not seem justified in view of the severe reaction to a relatively poorly absorbed form (see Comment).

COMMENT

It is of interest to consider why toxic reactions to ammoniated mercury are so rare as compared with the relatively frequent poisoning resulting from the use of antisyphilitic mercurial ointments. Unguentum hydrargyri (U.S.P.) and unguentum hydrargyri cinereum (Ph.G.). In this connection Maloff[11] reports absorption experiments with ammoniated mercury rubbed into the skin of volunteers. On the basis of urinalysis he concluded that only traces of mercury were absorbed. He cited the work of Bürgi[3] as reporting much greater absorption of mercury after the use of gray ointment (unguentum hydrargyri cinereum). Maloff believed that the rarity of toxic effects from ammoniated mercury was accounted for by its poor absorption and that this was due to its being a complex insoluble mercury compound, NH_2HgCl, in contrast to the better absorbed metallic mercury

mixtures of blue ointment and gray ointment. Moncorps[13] in a careful experimental study confirmed and greatly elaborated the work of Maloff, and Sollman[20] presents data on the absorption of unguentum hydrargyri.

In our cases it is suggested that the symptoms were manifestations of an idiosynerasy to mercury rather than the result of absorption of relatively large amounts of mercury. Presumptive evidence for this is found in the rapid development of symptoms after application of relatively small amounts of ammoniated mercury, in the experimental demonstration that mercury in this form is relatively poorly absorbed, in the rapid recovery following the withdrawal of the drug, in the absence of evidence of renal damage, and finally in the acute and severe reactions to skin tests.

SUMMARY

Local application of ammoniated mercury in two cases gave a generalized rash and stomatitis together with a peculiar cyanosis and edema of the hands and feet. It is believed that these signs and symptoms were the expression of an idiosynerasy to mercury.

REFERENCES

1. Alexander, A., and Mendel, K.: Deutsche med. Wchnschr. **49**: 1021, 1923.
2. Bechet, P. E.: Arch. Dermat. & Syph. **1**: 636, 1920.
3. Bürgi, Emil: Arch. f. Dermat. u. Syph. **79**: 81, 1906.
4. Cockle: Med. Times and Gaz., Lond. **1**: 302, 1882.
5. Deakin, S.: Brit. M. J. **11**: 1281, 1883.
6. Giles, J.: Lancet **11**: 9, 1857.
7. Graham, A. R.: Brit. M. J. **1**: 329, 1868.
8. Green, W. E.: Brit. M. J. **1**: 853, 1884.
9. Hardy, H. N.: Brit. M. J. **11**: 76, 1876.
10. Kerner: Med. Ann. Heidelb. **1**: 479, 1835.
11. Maloff, G.: Deutsche med. Wchnschr. **54**: 1381, 1928.
12. Michael, W. H.: Assoc. M. J. Lond. (now the Brit. M. J.) **11**: 909, 1857.
13. Moncorps, Carl: Arch. f. exper. Path. u. Pharmakol. **155**: 51, 1930.
14. Moore, E. H.: Brit. M. J. Lond. **11**: 15, 1885.
15. Newhall, W. A., and Prytherck, J. R.: Brit. M. J. p. 1684, 1900.
16. Personal communications.
17. Reynolds, E. S.: Brit. M. J. pp. 1492, 1520, 1590, and 1769, 1900.
18. Sandberg, A. G.: Brit. M. J. **1**: 709, 1889.
19. Short, A. C.: Med. Times and Gaz., Lond. **1**: 444, 1860.
20. Sollman, Torald: A Manual of Pharmacology, ed. 4, p. 1078, Philadelphia, 1932, W. B. Saunders Company.
21. Stevens, E.: Brit. M. J. **1**: 781, 1876.
22. Stevenson, T.: Guy's Hosp. Rep., Lond. 3. s. **19**: 415, 1874.
23. Symposium: Epidemic of Peripheral Neuritis Traced to Arsenical Contamination of Beer-Making Materials, Brit. M. J. pp. 1587, 1683, 1723, 1730, and 1815, 1900.
24. Taylor, E. S., and Pavy, F. W.: Guy's Hosp. Rep. **6**: 483, 1860.

EXANTHEM SUBITUM WITH ENCEPHALITIC ONSET

MARK J. WALLFIELD, M.D.
BROOKLYN, N. Y.

ENCEPHALITIC and meningeal syndromes have been noted in many instances, either ushering in or associated with an acute infectious process. Measles, mumps, varicella, whooping cough, vaccination, and diphtheria are common bases for a true encephalitis, as noted by S. A. K. Wilson.[1] Reports of involvement of the nervous system in exanthem subitum have been few, and the admission of a patient to the pediatric ward with the clinical picture of encephalitis and its subsequent dramatic change to one of exanthem subitum prompted this report. The age of the patient, twelve years, was also rather out of the ordinary.

CASE REPORT

B. K., male, aged twelve years, was admitted to the pediatric ward on Mar. 18, 1931, with a history of vomiting, fever, headache, vertigo and stiffness of the neck of two days' duration. A questionable convulsive seizure at home was also described. During the night his fever dropped, accompanied by sweating, but rose to 104.8° F. the following morning. No further vomiting or convulsions were noted, but since the headache, fever, and stiffness of the neck persisted, the child was hospitalized. His past history showed an attack of mumps, "two attacks" of measles, and frequent sore throats and grip. The family history was irrelevant.

Physical examination on admission revealed a sick child, somewhat stuporous, with a temperature of 105.4° F., pulse 110, and respirations 25. The conjunctivae and throat were slightly injected. Small, but tender, cervical lymph nodes were palpable. The heart and lungs showed no abnormalities. The liver and spleen were both palpable below the costal margin. Neurologically, moderate neck rigidity and Kernig's sign were present. The eyegrounds were negative, and no cranial nerve involvement was noted.

On the basis of the history of headache, fever and vomiting, and findings of a stiff neck and Kernig's sign, a tentative diagnosis of encephalitis was made, and a spinal puncture was made. The spinal fluid was clear and the pressure only slightly increased. Six cells per cubic millimeter were found; glucose was present; globulin, absent; and culture of the fluid was negative.

On March 20, two days later, the headache and neck rigidity disappeared, but the spleen was still palpable and somewhat tender. The Kernig sign was still positive. At this time a few faint, rose-colored spots were noted on the abdomen. After a pseudocritical fall of temperature to 99° F. during the night and a rise to 104° the next morning, a sudden drop in temperature to 100° F. took place. At the same time a rash appeared on the trunk more prominent posteriorly than anteriorly, consisting of pinkish red and bright rose red macules varying in size from a large pinhead to a split pea and disappearing completely on pressure. Within twenty-four hours the rash was present, on the extremities while that on the trunk

From the Pediatric Service of Dr. Henry Heiman and Dr. J. M. Wallfield, Israel Zion Hospital, Brooklyn, N. Y.

was beginning to fade. No desquamation was noted at any time. The temperature came down to a normal level over a period of two days; the rash was gone at this time, and the patient was discharged on the eighth day after admission.

Laboratory Examinations.—Throat culture, blood culture, and Widal reaction were negative. The urine showed only a faint trace of acetone. Blood counts showed red blood cells, 3,700,000; hemoglobin, 70 per cent Sahli; white blood cells ranged from 4,800 to 4,000; polymorphonuclears, 64 per cent; large lymphocytes, 29 per cent; small lymphocytes, 7 per cent.

SUMMARY

An uncommon case of exanthem subitum, which was ushered in by symptoms simulating encephalitis, is reported in a boy twelve years of age. After a high fever of three days' duration, a critical drop in temperature occurred, followed by a rash typical of exanthem subitum in its morphology, distribution, and duration with a rapid and uncomplicated recovery. The blood picture showed the characteristic leucopenia.

REFERENCE

1. Wilson, S. A. K.: J. Neurol. & Psychopath. 10: 36, 1929.

1650 Ocean Avenue

AURICULAR FLUTTER IN A NEWBORN INFANT

Case Report

Jules Sherman, M.D., and Robert A. Schless, M.D.
Philadelphia, Pa.

BEFORE entering into a discussion of the manifestations of our particular case, it is advisable to discuss briefly the symptom-complex of auricular flutter. This is a condition characterized by rapid, regular, and coordinated action of the auricles, in which the latter attain a speed so great that the ventricles are unable, or are rarely able, to respond to each auricular impulse, and hence a partial or a complete heart-block is set up. The impulses no longer arise from the sino-auricular node, but from some other portion of the auricle. The mechanism of the action is, according to Lewis' theory, as being due to stimuli passing over the auricular muscle in circus movement, so that the waves are contiguous to one another, and some group of muscle fibers is constantly in contraction. Usually there is a definite rhythm, indicating that there is present a regular number of auricular actions for each ventricular action. There may be two, three, four, etc., beats of the auricle to each one of the ventricle, but this may vary from time to time in the same patient. The term "auricular flutter," as applied to human auricles, was originally employed by Jolly and Ritchie in 1910.

From a pathologic standpoint it is claimed that any one of the following conditions may be present:

1. Acute or chronic inflammation of the endocardium or myocardium.
2. Infection of the pericardium.
3. Long-standing disease of the vascular system or cardiac muscles.
4. Disease of the conduction system (also generally of infectious origin).

In the laboratory, faradic stimulation of the auricular appendix will produce a flutter which is closely analogous to the auricular flutter at the bedside examination. Therefore, the statement has been made frequently that auricular flutter results from stimulation, excessive in frequency and intensity, of a localized area in some part of the auricular wall.

The diagnosis is made by comparing the jugular pulsation with the

From the Pediatric Service of Dr. Robert A. Schless, Jewish Hospital, Philadelphia.

pulse rate or by electrocardiogram. This shows a stream of regular P-waves which are intercepted every second or third beat by ventricular complexes of supraventricular type. The auricular-ventricular block may be of higher degree or of mixed type.

Carr and McClure[1] reported the first case of auricular flutter in a newborn infant in 1931. The patient was a full-term, ten-pound baby and was examined six minutes after birth. At this time the heart rate was about 180. Twenty-six hours later diagnosis by electrocardiogram showed auricular flutter. The heart returned to a normal rhythm ten days after birth, and at no time since has there been a recurrence of the abnormality. This is the only case of auricular flutter in a newborn infant that we could discover in a review of the literature. Several cases have been reported in infants and children.

Poynton and Wyllie[2] reported a case in 1926, which showed auricular flutter in a child of eight years. This was substantiated by electrocardiogram. They believed that their case was congenital in origin because the symptoms described in the first few weeks of life only differed in degree from those which brought the child to the hospital. To quote Poynton and Wyllie: "This child continuously showed a ventricular rate of 150 and is well grown, bright colored and as wayward as any healthy child of corresponding age." A case such as this, well illustrates the remarkable powers of the natural processes of growth and development to assert themselves, despite the grave anomalies in the circulation. The child overcame an attack of bronchopneumonia as readily as a normal child.

Hume in 1913 reported two cases of auricular flutter following diphtheria. In one of these the auricular rate reached 500 with a 5 to 1 rhythm of the auricle to the ventricle.

O'Donovan[3] in 1929 cites a case of a five-year-old boy who showed auricular flutter with an auricular rate of 330 and ventricular rate of 165. This followed an attack of diphtheria. Fortunately a normal rhythm was resumed in three days. He makes the following statement: "Flutter in childhood, even apart from diphtheria, is rare. In the records of the National Hospital for Diseases of the Heart no case can be found in a child so young as here described."

In a series of flutter cases published by Parkeson and Bedford, the youngest was twelve years old.

Amberg and Willius[4] in 1926 reported a case showing auricular flutter in a five-year-old child. The auricular rate was 330 and the ventricular 105. Their findings indicate "that digitalis will diminish the ventricular rate, but in no way affect the auricular rate." They make this pertinent comment: "Many attempts have been made to correlate auricular flutter with histopathologic findings in the auricle, but as yet no uniform or convincing association has been demon-

strated. Until more adequate proof is available, we must be content to accept the view that flutter occurs as the result of some physiologic disturbance of auricular function."

Obviously, the causes given are insufficient and practically all com-

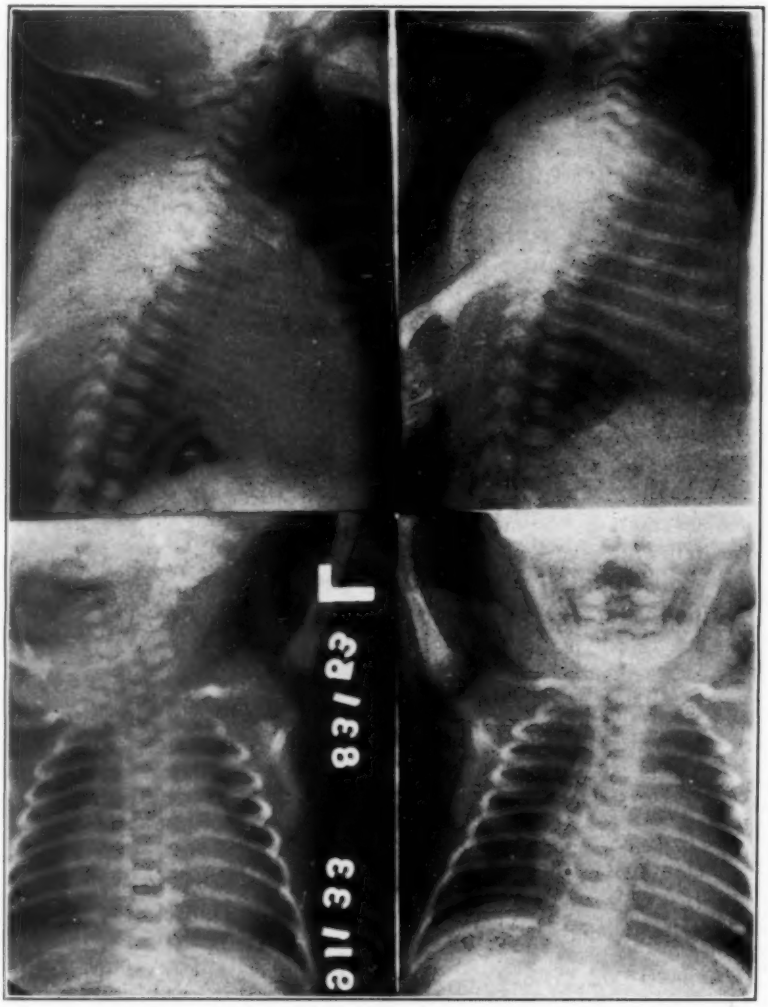


Fig. 1.—Right border, 2.4 cm. from midvertebral line; left border, 3.7 cm. from midvertebral line; inside measurement of chest wall, 8.5 cm. A normal heart would have a transverse measurement of 4.3 cm.; the measurement in our patient was 6.1 cm.

ments to date beg the question, in that they are comprised merely of influences of possible damage to auricular function. Maturity of the conduction bundle may be a late uterine development and may simu-

late true nervous tissue which it imitates, rather than the muscle of which it is part. Our conjecture concerning the basic cause of auricular flutter in a newborn infant is the underdevelopment of the conduction bundle tissue.

CASE REPORT

History.—J. R., a three-month-old boy was admitted to the Jewish Hospital Dec. 30, 1933, with occasional syncope and cyanosis.

During the eighth month of pregnancy, the obstetrician attending the mother

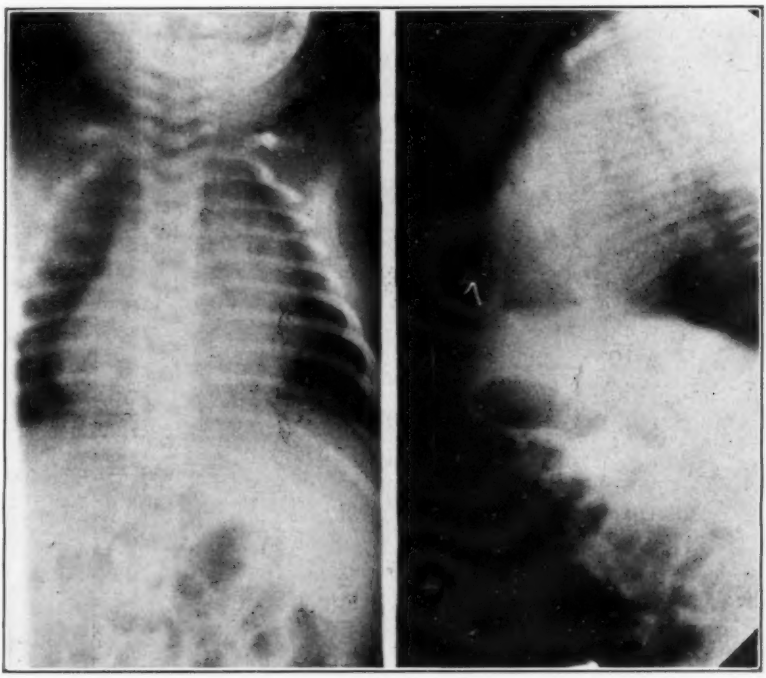


Fig. 2.—Right border, 3.3 cm. from midvertebral line; left border, 3.8 cm. from midvertebral line; inside measurement of chest wall, 11.2 cm. A normal heart would have a transverse measurement of 6.1 cm.; in our patient the measurement was 7.1 cm.

remarked that it was difficult to count the baby's heart beats because of the rapidity. Two weeks later, the same doctor prognosticated upon examination that the baby would be female since the fetal heart rate was 190. Both of these statements are significant in view of subsequent developments.

The mother was delivered of a seven-and-one-half-pound boy on Sept. 9, 1933. Outlet forceps were used, with the mother under nitrous oxide anesthesia. Immediately following birth, oxygen was administered to the infant because of improper breathing. At this time no notice was taken of cyanosis of the lips, skin, finger or toe nails. When the infant was four days old, a pediatrician was called in attendance. The child's nutrition was excellent, he gained weight; and his digestive system appeared normal. A routine x-ray picture of the thymus was re-

ported normal. Subsequent examination of the films revealed moderate cardiac hypertrophy (Fig. 1). One month later the baby's breathing became so difficult that a specialist was called in. There was a decided blueness of the toe nails. The cardiac apex rate was 256. The prescription, mii tr. digitalis b.i.d., was continued for two weeks, resulting in little change. An electrocardiogram was taken two weeks later, and an auricular flutter was discerned. The auricular rate was 464 and the ventricular 232 (Fig. 3). At this time the x-ray examination of the chest was reported as follows (Fig. 2):

"The cardiac silhouette is increased in size. There is straightening of the left auricular border. The right heart is larger than the left heart. There is no buckling of the trachea. The diaphragms are smooth and sloping. The trachea is displaced to the right, and since the last examination of the anterior posterior view, there may be a lateral enlargement of the right lobe of the thymic gland."

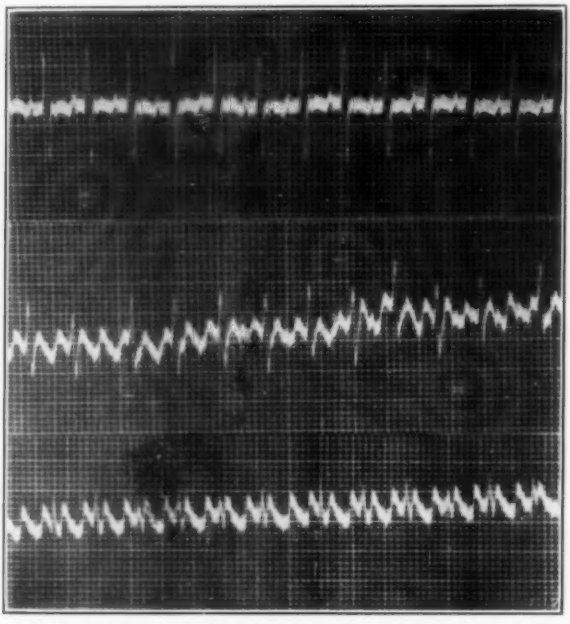


Fig. 3.—Electrocardiogram. Auricular rate, 464; ventricular rate, 232.

The patient received no treatment for enlarged thymus, and later this gland apparently returned to normal size spontaneously. Subsequent examination showed the heart to be greatly enlarged.

At the age of two months, the infant developed a cough from exposure. This additional symptom aggravated both the dyspnea and cyanosis. The mother noticed then that the infant was subject to "spells," which consisted of facial pallor, apparent weakness, sometimes so pronounced that the child was in a state of syncope. There was a positive blueness of the lips. These "spells" were evident every time the infant was fed and also accompanied each cough. Our deduction was that the act of swallowing caused a temporary cessation of breathing, more prolonged than normal, which produced an intensification of the current cardiac symptomatology.

A roentgenogram and an electrocardiogram made at the age of three months are shown in Figs. 4 and 5, respectively.

Family History.—Father, thirty-three years old, was living and has been consistently healthy. Mother, twenty-six years old, was told by her parents that when she was a child, a doctor ascribed a "heart murmur" to her. At present she suffers from gallbladder disease; the gallbladder is drained frequently. There is one other child, a boy, four years old, living and well.

Present Medical History.—Upon admission to the hospital, the infant was found to have cyanosis of the finger and toe nails. Difficulty in breathing was present but not as a pronounced symptom. During the patient's stay in the hospital, at no time

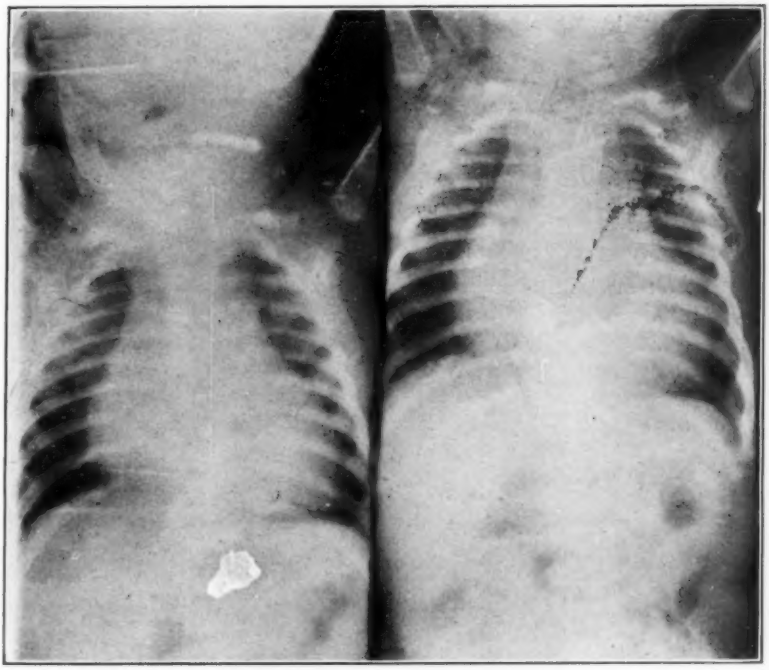


Fig. 4.—X-ray picture of the heart. Right border, 4.1 cm. from midvertebral line; left border, 5.0 cm. from midvertebral line; inside measurement of chest wall, 13.0 cm.; A normal heart would have a transverse measurement of 6.5 cm.; in our patient the measurement was 9.1 cm.

did he have an attack of syncope such as he had suffered at home. He had an upper respiratory infection.

Physical Examination.—We found a well-nourished, normally developed white infant showing evidence of distinct cyanosis and moderate dyspnea. Examination showed, in addition to an acute mild coryza, the heart to be the only apparent abnormality. The heart sounds over the precordium were extremely rapid, distinct, and normally rhythmic, except for an occasional irregularity. The sounds were similar in quality and intensity over the entire area of cardiac dullness. Percussion revealed a moderately enlarged heart, reaching to the right about one-half inch beyond the sternal border and to the left one-half inch beyond the nipple line. Exam-

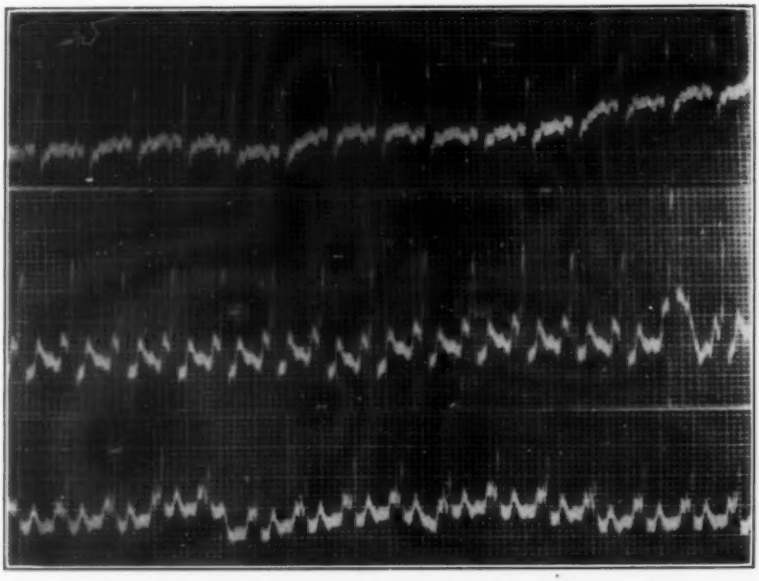


Fig. 5.—Electrocardiogram. Auricular rate, 420; ventricular rate, 210.

Fig. 6.—X-ray picture of the heart. Right border, 3.1 cm. from midvertebral line; left border, 5.1 cm. from midvertebral line; inside measurement of chest wall, 13.6 cm. A normal heart would have a transverse measurement of 6.8 cm.; in our patient the measurement was 8.2 cm.

ination of the x-ray picture taken Dec. 21, 1933, will reveal that our clinical findings were underestimated (Fig. 4).

Course.—The patient was given four drops tr. digitalis t.i.d. from Dec. 30, 1933 to Jan. 7, 1934; 5 drops haliver oil with viosterol, b.i.d.: sodium bicarbonate enema daily; ephedrine 1 per cent—neosilvol 10 per cent, 2 drops t.i.d., intranasally; and atropine sulphate $\frac{1}{600}$, if necessary, for cyanosis.

His formula consisted of:

Evaporated milk	11 oz.
Water	12 oz.
Karo syrup	1 oz.
	(4 bottles, 6 oz. each.)

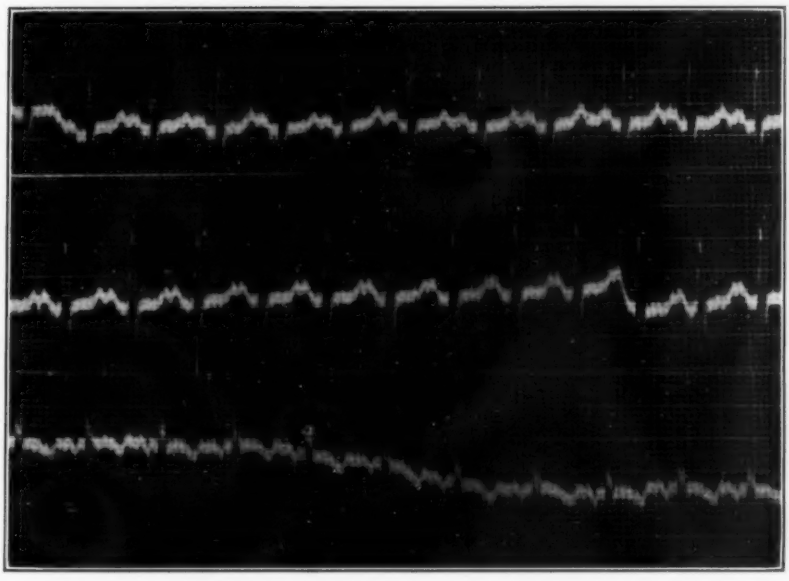


Fig. 7.—Electrocardiogram. Rate, 147; P-R interval, 0.12; QRS, slurred; P- and T-waves, inverted in Lead III.

Orange juice, 1 oz., was given daily, and cereal, 1 oz., at 10 A.M. and 1 oz. at 6 P.M.

The pulse rate receded as follows:

Dec. 30, 1933	220
Dec. 31, 1933	120
Jan. 4, 1934	150
Jan. 8, 1934	146
Jan. 10, 1934	150

The sudden decrease in the heart rate may be attributed to the tincture of digitalis that was given. The cyanosis and dyspnea gradually diminished and were gone entirely when the patient was discharged on Jan. 11, 1934.

At home the infant acted and appeared perfectly normal in every respect. February 6 the patient was returned to the hospital for a roentgenogram and an electrocardiogram, the results of which are shown in Figs. 6 and 7, respectively.

COMMENT

As closely as we can determine, this case is the second to be reported in the literature. We believe that the auricular flutter was present at birth for the following reasons:

1. During the eighth month of pregnancy the heart rate was so fast that it could not be counted.

2. The obstetrician's statement that he believed the infant would be female, because the fetal heart rate was 190.

3. The greatly enlarged heart shown by an x-ray picture taken at two days of age.

4. Cyanosis of the finger nails noted at four days of age.

We believe that the maturity of the conduction bundle established the normal rhythm.

REFERENCES

1. Carr, J. G., and McClure, W. B.: Am. Heart J. **6**: 824, 1931.
2. Poynton, F. J., and Wyllie, W. G.: Lancet **2**: 371, 1926.
3. O'Donovan, P.: Lancet **1**: 607, 1929.
4. Amberg, S., and Willius, F. A.: Am. J. Dis. Child. **99**: 104, 1926.
5. Keating, J. H., and Hajek, J.: Am. J. M. Sc. **164**: 656, 1922.

3035 West Diamond Street

AURICULAR FIBRILLATION AS AN EARLY TOXIC DIGITALIS MANIFESTATION

Further Observations on This Drug in Children With Congestive Heart Failure

Abraham Jezer, M.D., and Sidney P. Schwartz, M.D.
New York, N. Y.

IN RECENT studies on children with rheumatic fever and normal sinus rhythm with signs of congestive heart failure, we observed that the administration of digitalis resulted in a change from normal sinus rhythm to auricular fibrillation in two instances. In one child auricular fibrillation was preceded by the appearance of auricular premature beats which were recorded electrocardiographically. In the second child the arrhythmia appeared after the onset of nausea and vomiting. The irregular rhythm in both children was transient and disappeared after the withdrawal of the drug.

Similar instances in the change of the heart rhythm from normal to auricular fibrillation have been described by Neuhof.[1] In a child, eight years old, with rheumatic heart disease to whom digitalis was administered with the hope of diminishing the congestive heart failure, auricular fibrillation appeared on three separate occasions following the administration of small quantities of the drug. With the withdrawal of digitalis normal sinus rhythm returned each time.

In 1913 a somewhat similar experience was described by Nadel,[2] and we observed several instances which have already been reported.[3, 4] However, these observations have been considered by some to be fortuitous, and although some have concurred in the view that digitalis may initiate auricular fibrillation in children,[5, 6] there still exists some skepticism about this possibility. It is for this reason that we have thought it worth while to call attention to the limitations in the use of digitalis in children with rheumatic fever and congestive heart failure.

In this study, tincture of digitalis was administered to seven children in daily doses of 2 c.c. (1 c.c. is the equivalent of 1 cat unit) in the hope of diminishing the congestive heart failure. All the children had rheumatic valvular heart disease, and each had suffered two or more recurrences of active rheumatic fever. This study was carried

From the Medical Division of the Montefiore Hospital, service of Dr. Leopold Lichtwitz.

on during a period when the children were afebrile. The heart rates, rhythm, and weights were recorded daily, and the signs of congestive failure were observed. The children received a regular diet with the fluids restricted to approximately 900 c.c. a day (including the fluid in the diet). They received no other diuretic therapy during the control period of six weeks and their daily weights did not fluctuate more than two or three pounds during this period. They were kept in bed during the control period and during the period of this study. The detailed case reports of two cases in whom changes in rhythm occurred are as follows:

CASE 1.—W. A., an eleven-year-old boy, was well until his eighth year when he suffered an attack of migratory polyarthritis. A diagnosis of heart disease was made soon following this, and congestive heart failure appeared at the age of nine, when he was admitted to the Montefiore Hospital. Following a period of rest in bed and restriction of fluids, the signs of congestive heart failure disappeared. He was discharged and remained at home free from any symptoms until April 7, 1931, when he was readmitted because of vague pains and aches.

He was pale, and his neck veins were slightly distended. The left nipple was displaced upward and outward. The apical impulse of the heart was in the sixth intercostal space in the anterior axillary line. A loud blowing diastolic murmur could be heard over the left fourth intercostal space near the sternal border. The heart rhythm was regular, and the rate averaged 80 beats per minute. The lungs were clear. The liver edge was felt 8 cm. below the right costal border in the right midclavicular line. There was some edema of the lower extremities and face.

On May 22, 1931, he received his first dose of 2 c.c. of the tincture of digitalis. On each subsequent day at 9:00 A.M. the same quantity of the drug was given. The patient remained afebrile and free from symptoms. The heart rate was slightly slower, but the liver enlargement did not diminish, and the body weight ranged from 71¾ pounds on May 26, 1931, to 70¼ pounds on July 16, 1931, when the drug was withdrawn. On July 16 he began to complain of transient palpitations of the heart. These were frequently accompanied by precordial pain. Examination at this time revealed the rhythm of the heart to be totally irregular for a few minutes; then the heart action became regular again except for an occasional premature beat. At times the premature beats alternated with regular sinus beats when a bigeminal rhythm was heard on auscultation (Fig. 2).

An electrocardiogram (Fig. 1) taken at the onset of the complaints showed a sinus rhythm interrupted by premature auricular beats. There was also a marked sinus arrhythmia. The P-R intervals were within normal limits. The heart rate varied between 100 and 160 beats per minute, and the rhythm was totally irregular. The next day he complained of palpitations, but otherwise he was comfortable. Electrocardiograms taken at this time revealed a dominant sinus rhythm interrupted by many recurrent premature auricular beats (Fig. 3), but no pulse deficit was present. Later that day the patient complained of increased palpitations, and on auscultation no period of regularity of the heartbeat could be heard; a pulse deficit was present. An electrocardiogram at this time revealed the presence of auricular fibrillation (Fig. 4). On July 18, 1931, the irregular rhythm persisted, the patient became dyspneic, the size of the liver increased, and later that day he was nauseated and vomited. On July 19, 1931, the heart rate varied between 140 and 170 beats per minute with a pulse deficit of from 30 to 40 beats per minute. The signs of

Fig. 1.

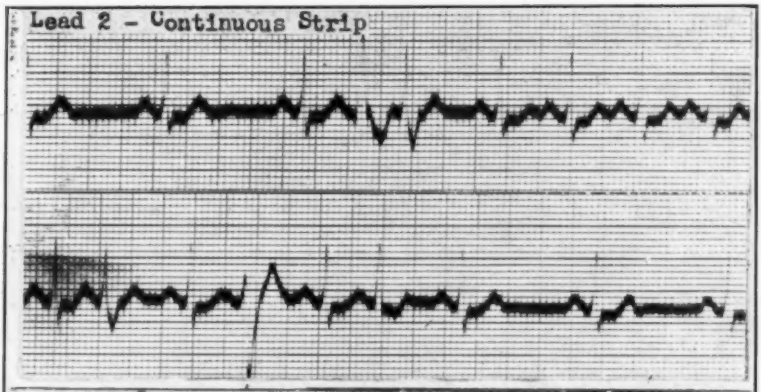


Fig. 2.

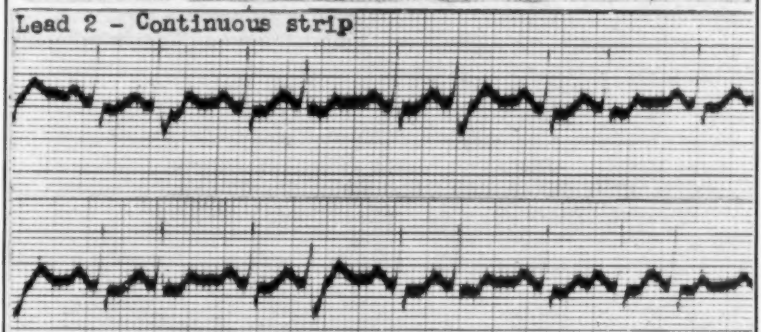


Fig. 3.

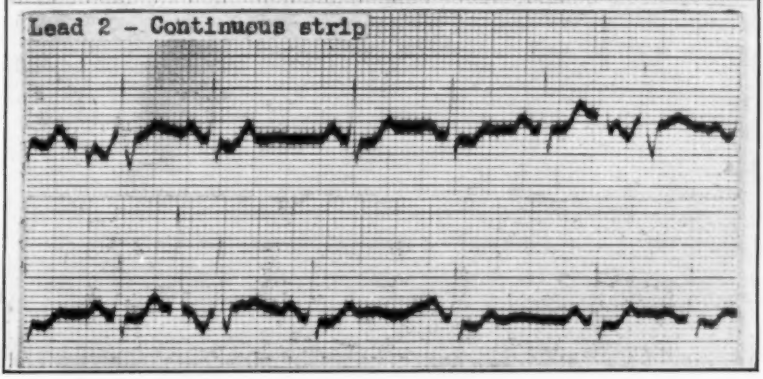


Fig. 1.—(Case 1, W. A. July 16, 1931.) Sinus arrhythmia interrupted by premature beats of auricular origin, following the administration of 2 cat units of digitalis daily for fifty-eight consecutive days. No other signs of digitalis intoxication. Digitalis discontinued.

Fig. 2.—(Case 1. July 16, 1931.) Bigeminal rhythm due to alternate premature beats of auricular origin.

Fig. 3.—(Case 1. July 17, 1931.) Multiple premature beats of the auricle with a slower ventricular rate.

congestive failure had increased; moist râles appeared in the lungs, and the liver enlargement became still greater. On July 20, 1931, the heart rhythm suddenly returned to regular rhythm, the heart beating at the rate of 125 beats per minute.

Fig. 4.

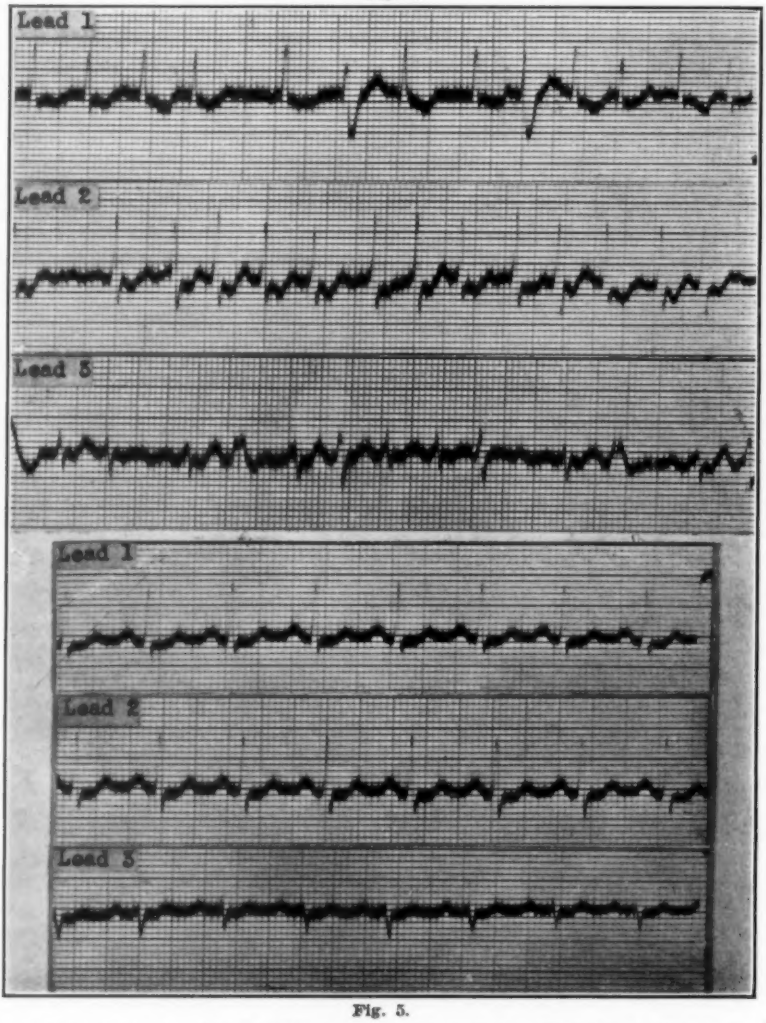


Fig. 5.

Fig. 4.—(Case 1. July 17, 1931.) Transient auricular fibrillation lasting three days. Onset one day after discontinuance of digitalis. Ventricular rate ranged from 140 to 170 beats per minute. Pulse deficit of from 30 to 40 beats per minute.

Fig 5.—(Case 1. July 20, 1931.) Return to sinus rhythm. Ventricular rate of 120 to 125 beats per minute.

Electrocardiographic evidence of the return to regular sinus rhythm was obtained, the patient was more comfortable, and the dyspnea was definitely improved (Fig. 5).

He was free from palpitations, the nausea and vomiting disappeared, and within one week following the return to sinus rhythm, the increasing signs of congestive heart failure had subsided.

COMMENT

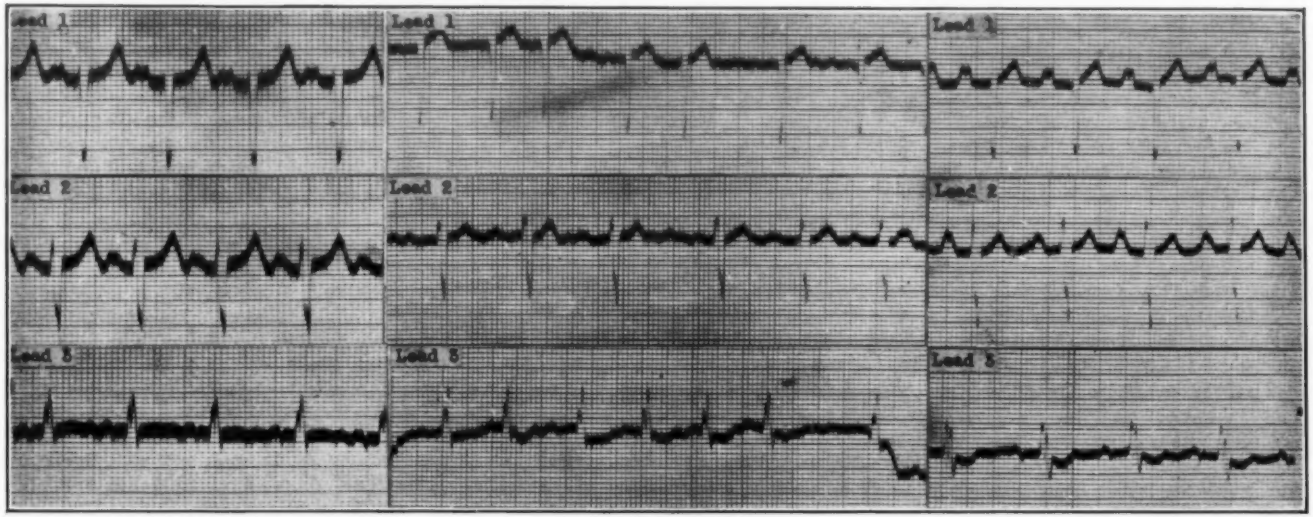
A daily dose of digitalis equivalent to 2 cat units was given for fifty-eight days to a boy who had rheumatic heart disease and congestive heart failure, but there was a definite change in the rhythm of the heart. Auricular premature beats were noted as the first toxic effects of the drug. Although the drug was stopped at this time, its cumulative effects were so great that on the following day auricular fibrillation developed. Nausea and vomiting, usually described as an early manifestation of digitalis toxemia, was not present until the signs of congestive failure had increased, following the onset of auricular fibrillation. This irregular rhythm disappeared after three days.

It can be seen: (1) that nausea and vomiting in this instance were not early signs of digitalis toxemia; (2) that auricular premature beats were the first noted toxic effects of this drug; and (3) that auricular fibrillation followed soon after the onset of the recurrent auricular beats.

CASE 2.—R. B., a four-year-old girl, was admitted to the Montefiore Hospital Mar. 27, 1930. At the age of three she suffered from recurrent sore throats which disappeared following tonsillectomy. In January, 1930, swelling of the abdomen was noted, and the patient was admitted to the Jewish Hospital of Brooklyn. A diagnosis of rheumatic heart disease, mitral stenosis and insufficiency, and moderate congestive heart failure was made.

Examination on admission to the Montefiore Hospital three months later showed the child to be well nourished with a marked malar flush and cyanosis of the lips. The cervical neck veins were distended. The left nipple was displaced upward and outward. There was a marked bulging of the anterior chest wall in the region from the second to sixth left costosternal junctions. The apical impulse of the heart was felt and heard in the sixth intercostal space in the anterior axillary line. The first heart sound was accentuated and partly obscured by a blowing systolic murmur. P_2 was accentuated. The lungs were clear. The liver was markedly enlarged, its edge being felt 10 cm. below the right costal border in the right midclavicular line. There was edema of the lower extremities.

The patient was febrile for several months, following which the rheumatic activity subsided, and signs of congestive heart failure diminished. The liver edge remained at the umbilical level. The child was no longer dyspneic at rest although slight cyanosis of the lips was still present. She was afebrile during the spring of 1931 and on May 26, 1931, and on each following day until June 9, 1931, at 9:00 A.M., she received 2 c.c. of the tincture of digitalis. The weight on the first day of the administration was 40 pounds, and on the last day it was 37 pounds. She was afebrile throughout this course. She had no complaints throughout the period of administration of the drug until June 9, when she became nauseated and vomited. The drug was discontinued at this time. Later that day she complained of palpitations, and examination revealed that the rhythm of the heart had become irregular. There was a pulse deficit of from 10 to 20 beats per minute. Electrocardiogram revealed a totally irregular heart rhythm with fibrillation of the auricles and a ventricular rate of 100 beats per minute. The R-T_1 take-off was high (Fig. 6-*B*).



A. *B.* *C.*

Fig. 6.—(Case 2, R. B.) *A.* Before digitalis was administered. P-R interval: 0.18-0.20 seconds.
B. Transient auricular fibrillation lasting nineteen hours, following the administration of 2 cat units of digitalis daily for fifteen days. Digitalis discontinued.
C. Return to sinus rhythm on the following day. Sinus arrhythmia. P-R interval: 0.24-0.32 second.

The irregular rhythm lasted for about nineteen hours and then suddenly became regular. Electrocardiograms at this time revealed a normal sinus rhythm (Fig. 6-*C*). The P-R interval ranged from 0.24 to 0.32 seconds. There was a negative T_3 and a marked sinus arrhythmia. The rate was 105 beats per minute.

COMMENT

A daily dosage of digitalis equivalent to 2 cat units was given for fifteen days to a girl who had rheumatic heart disease and congestive heart failure. There was no apparent change in the signs of congestive failure. Nausea and vomiting was followed by the change from sinus rhythm to auricular fibrillation. The auricular fibrillation lasted for nineteen hours, and the electrocardiogram following the return to sinus rhythm showed an increased P-R interval and a marked sinus arrhythmia which are typical electrocardiographic changes produced by excessive digitalis action upon the heart.

CASES 3 TO 7.—The other five children to whom the drug had been administered were all similar to the two cases described. All had rheumatic heart disease and from mild to moderate congestive heart failure. The digitalis was administered in 2 c.c. (2 cat units) doses over a period of fifty-eight days. There was no improvement in the congestive heart failure. The heart rate, which ranged from 90 to 110 beats per minute before the drug was administered, was only slightly lowered following the administration of the drug. No clinical or electrocardiographic evidence of digitalis intoxication was noted.

DISCUSSION

The continued administration of digitalis bodies over a prolonged period of time may not induce nausea and vomiting as an early sign of excessive digitalis administration. In one case reported in this communication, auricular fibrillation followed the onset of nausea and vomiting. In another case, premature beats of the auricle and fibrillation of the auricles preceded the onset of nausea and vomiting as the first clinical manifestations of digitalis toxemia.

The value of slowing the heart action, we believe, is an important one, since it reduces the heart work. We would favor the use of the drug in such cases if this effect were usual and not accompanied by signs of digitalis intoxication. In our experience with the drug, however, the slowing of the heart action in the presence of sinus rhythm is usually not great, and the incidence of digitalis poisoning not infrequent where adequate therapeutic doses of digitalis have been administered. We have also observed the consistent therapeutic failure of digitalis and the successful therapeutic use of diuretic measures in cases of moderate congestive heart failure.

SUMMARY AND CONCLUSIONS

1. Seven children with rheumatic heart disease and congestive heart failure were given 2 cat units of digitalis daily over a period varying from fifteen to fifty-eight days. Auricular fibrillation was an early toxic

manifestation of this drug in two cases. In the first case premature beats of the auricle preceded the onset of auricular fibrillation. In the second case nausea and vomiting preceded the irregular rhythm by one day.

2. Daily doses of 2 cat units of digitalis given over a period of from fifteen to fifty-eight days did not produce diuresis in seven children who had rheumatic heart disease and moderate congestive heart failure in the presence of regular sinus rhythm.

REFERENCES

1. Neuhof, S.: Clinical Cardiology, New York, 1917, The Macmillan Co., p. 63. Med. Clin. North America **4**: 307, 1920.
2. Nadel, V.: Mitt. d. Gesselsch. f. inn. Med. u. Kinderh. in Wien **12**: 210, 1913
3. Schwartz, S. P., and Weiss, M. M.: Am. J. Dis. Child. **36**: 22, 1928.
4. Schwartz, S. P.: Am. J. Dis. Child. **39**: 549, 1930.
5. Cookson, H.: Lancet **2**: 1139, 1929.
6. McEachern, D., and Baker, B. M.: Am. J. M. Sc. **183**: 35, 1932.

FLUOROSCOPICALLY CONTROLLED ENEMA REDUCTION OF INTUSSUSCEPTION

E. D. Huntington, M.D., and R. E. Williams, M.D.
Chicago, Ill.

THE possibility of nonoperative reduction of ileocolonic intussusceptions by enema has recently been recalled to the medical mind by a publication of Edwin Miller,[1] in which he reviews the work of Hipsley[2] of Australia. The frequent success of this ancient procedure has been largely lost sight of in this day of surgically minded practitioners. Various writers have condemned the procedure as "unscientific," of "historical interest only," and as "dangerous" and "uncertain."

Hipsley in 100 consecutive cases was able to reduce sixty-two by pressure enemas, using saline solution, with the patient anesthetized; of these sixty-two cases he found it necessary to verify the reduction by laparotomy in eighteen cases. His mortality for the entire series of 100 cases was 5 per cent.

Retan[3] in 1927 suggested reduction by fluoroscopically controlled enema technic. An essentially similar method was independently described by Stephens[4] the following year. Each writer reported a successfully reduced case. The advantages claimed for the fluoroscopic method by these writers are safety, anesthetic unnecessary, and certain recognition of success or failure.

The method described by both Retan and Stephens is briefly as follows: The barium mixture is introduced with minimal pressure until the colon is filled to the advancing point of the intussusceptum (the enema source not being higher than 3 feet above the table); then with the gravity pressure shut off, the barium is forced against the intussusceptum by manual pressure externally on the already filled colon under visual guidance and control. As the intussusceptum recedes more of the mixture is introduced and manual pressure again applied. As soon as the intussusception seems reduced, the colon is completely filled by gravity pressure to make certain the reduction is complete; if such is the case the colon fills completely and the barium mixture can be seen passing into the terminal ileum. These fluoroscopic observations are checked by an immediate roentgenograph.

Retan lists the following points as evidence of complete disinvagination: (1) The colon will fill completely with leak into the ileum as

evidenced by fluoroscopic view and confirmed by roentgenograph; (2) the abdominal tumor disappears; and (3) the child is relieved of his symptoms, shock, pain, vomiting, etc.

We believe this method has several advantages. It is intelligent, scientific, safely conservative, and analogous to the conservative and proper conduct of labor as compared to routine abdominal section. Diagnosis is definitely established even if the procedure fails. Visual control makes for effectiveness, safety, and certainty as to success or failure. An anesthetic is unnecessary.

If reduction of the intussusception fails no harm is done, and appropriate surgical treatment can be promptly instituted. If only partial success is obtained, the procedure is still well worth while, since in the opened abdomen it is far simpler to deal with a mass that extends no further than the hepatic flexure than with one which extends to the sigmoid portion of the colon.

Retan has advised that this method be used only within the first twelve hours of the intussusception, on theoretical safety considerations. But the degree of severity (extent, agglutination, edema, strangulation, and lessened vitality) certainly does not depend on the time element alone. And, moreover, who can determine how long the condition has been present in a given case? Usually one can only know how long the patient has had symptoms of gastrointestinal distress, which often antedate, sometimes for days, the actual occurrence of the intussusception. Hipsley fails to state the criterion by which he determined how long the actual intussusception had existed in his patients; other writers are equally indefinite.

We present briefly the case history of a successfully reduced intussusception in which a fluoroscopically controlled barium enema was used, with roentgenographs taken before and after reduction.

Robert F., aged two years, was seen on Sept. 26, 1933, because of severe pain in the abdomen. This pain had begun at about 5 A.M. and recurred at frequent intervals during the next two hours. Vomiting occurred at 7 A.M. while one of us was examining him; shortly after a spontaneous normal bowel movement occurred. There was a palpable and visible mass in the right lower quadrant of the abdomen; it was sausage shaped and about 2 inches long. The mass was tender, but the rest of the abdomen was of normal sensitivity, and there was no rigidity. The child was sent to the hospital with a tentative diagnosis of acute intussusception.

On admission to the hospital the temperature was 98.8° F. (rectal); respirations, 26; and pulse, 130. Atropine sulphate, gr. $\frac{1}{500}$, was given hypodermically, and the child sent to the x-ray division at 9 A.M. At this time the tumor mass was in the epigastrium. A thin barium enema was given with the double purpose of checking the clinical diagnosis and of attempting pressure reduction of the invagination. The examination confirmed the diagnosis (Fig. 1), but the enema failed to reduce the invagination while being observed under the fluoroscope. The child expelled a large part of the enema soon after the rectal tube was removed, and the nurses commented that he seemed relieved of all pain. On return to the ward, the child

Fig. 1.—The enema readily distended the colon until it came to the site of the tumor mass, at which point it was totally obstructed, although it "pushed" the intussusceptum back to the right upper quadrant. Fine lines of opacity soon extended past the point of initial obstruction, evidently representing "seepage" about the invaginated portion, i.e., infiltration between the opposing surfaces of the invaginated and receiving portions of the gut, respectively.

Fig. 2.—The enema has completely filled the colon, and there is some passage into the ileum.

fell asleep, and on palpating the abdomen it was found that the tumor mass had disappeared. Re-examination under the fluoroscope with a barium enema showed the colon filling normally (Fig. 2).

The child's previous history was normal. Physical examination revealed no abnormalities other than those noted above. There has been no recurrence of this condition to date.

One of us (E. D. H.) has had occasion to see three other successful reductions by enemas of clinically typical intussusceptions. In all three cases the pathology had existed but a few hours.

In the last two years in two infants attempted reduction by enema under the fluoroscope was unsuccessful; in one the condition had been presumably present over three days, and in the other over one day. Both cases were with difficulty reduced at operation.

It is our impression that the medical publications, especially those reaching the general practitioner, have neglected to keep the profession conscious of this grave condition occurring in infancy and childhood. Too often the diagnosis is not made until twenty-four or more hours have elapsed; bloody stools are too often the first sign to arouse the attendant from his lethargy. Flat roentgenographs of the abdomen would be helpful even in intussusception of the small intestine; if the colon is involved, opaque enema is diagnostic from the first.

If the diagnosis is made early, fluoroscopically controlled enema offers a good chance of nonoperative reduction in ileocolonic invagination; if it fail or if the invagination be the small intestine, early operative reduction is available at an optimum time.

REFERENCES

1. Miller, Edwin: Ann. Surg. 48: 706, 1933.
2. Hipsley, P. L.: M. J. Australia 2: 201, 1926.
3. Retan, G. M.: Am. J. Dis. Child. 33: 765, 1927.
4. Stephens, V. R.: Am. J. Dis. Child. 35: 61, 1928.

1357 West 103rd Street

Pediatric Clinics

THE BABIES AND CHILDRENS HOSPITAL OF CLEVELAND AND THE DEPARTMENT OF PEDIATRICS, SCHOOL OF MEDICINE, WESTERN RESERVE UNIVERSITY

HENRY J. GERSTENBERGER, M.D.*
CLEVELAND, OHIO

THE department of pediatrics of the School of Medicine of Western Reserve University has its principal activities in the Babies and Childrens Hospital,† which is one of the University Hospitals of Cleveland, and coordinate activities at the Cleveland City Hospital and at the Rainbow Hospital for Crippled and Convalescent Children, which is also a unit of the University Hospitals. A brief statement with regard to the organization of the department will show how the activities are correlated. The professor of pediatrics at the School of Medicine is the director of pediatrics of the Babies and Childrens Hospital and also the head of the departments of contagious diseases and of pediatrics at the Cleveland City Hospital and director of pediatrics at the Rainbow Hospital; he is also director of pediatrics at the out-patient departments of the University Hospitals and of the Cleveland City Hospital. At the Cleveland City Hospital Dr. John A. Toomey is in active charge of the department of contagious diseases and Dr. J. D. Pilcher, of the department of pediatrics. Dr. Pilcher is also in charge of the East Thirty-Fifth Street Dispensary, which was formerly operated by the Babies' Dispensary and Hospital and is now a part of the out-patient department of the Cleveland City Hospital. The teaching staff of the department of pediatrics is made up of "full-time," "part-time," and "voluntary" physicians who are active in these institutions and in other affiliated agencies which will be referred to later.

HISTORY OF THE BABIES AND CHILDRENS HOSPITAL

The present building of the Babies and Childrens Hospital of Cleveland was opened during December, 1925. The activity culminating, finally, in the erection of this building had been of vital service in Cleveland since July, 1906. The name "Babies and Childrens Hospital" was taken in 1925 at the time of the erection of the present building in order to indicate by this name that the scope of this institution had been increased to include the care of children as well as that of infants.

In July, 1906, the Infants' Clinic of the Milk Fund Association and of the Visiting Nurse Association was organized at the suggestion of Dr. John H. Lowman, professor of medicine of Western Reserve University, a physician who was intensely

*Director of Pediatrics, Babies and Childrens Hospital, and Professor of Pediatrics, School of Medicine, Western Reserve University.

†In April, 1933, the Board of Trustees of the University Hospitals, because of marked reduction in income from endowment funds, from the Community Fund, and from patients at the University Hospitals as a whole, decided to reduce temporarily the number of beds in medicine, surgery, obstetrics, and pediatrics. In order to do this most economically, the two smaller units were closed, namely, the Babies and Childrens Hospital and the Maternity Hospital, and were given space vacated by the departments of medicine and surgery in the new Lakeside Hospital.

interested in the problem of tuberculosis and who appreciated keenly the importance of the social aspect of medicine in general. Dr. Lowman realized that by bringing together the services of the Milk Fund Association and of the Visiting Nurse Association and the special training of the present director of the Babies and Childrens Hospital, who at that time had just returned from a two-and-one-half-year period of study in Germany, the infant welfare work which had since 1902 been carried on during the summer months in Cleveland could be enlarged, intensified, and modernized. The work of this clinic fortunately received the hearty approval of Dr. Edward F. Cushing, professor of diseases of children of Western Reserve University, who had for some time been much interested in having established in Cleveland a hospital similar to the Babies' Hospital in New York. As a result of Dr. Cushing's efforts the Infants' Clinic was reorganized as the Infants' Clinic and Hospital, and the plans were enlarged to include the creation of a hospital. Under the stimulus of Dr. Cushing's enthusiasm funds were raised and sufficient property purchased on Longwood Avenue, later known as East Thirty-Fifth Street, to make possible the erection of a hospital building, a dispensary building, a nurses' home, and a milk laboratory. On April 1, 1907, the present director of pediatrics was invited to become the full-time medical director of the Babies' Dispensary and Hospital, as the new organization was then named. Inasmuch as sufficient funds for the immediate realization of this comprehensive plan were not forthcoming, the interest and energy of the new organization were entirely directed toward improving the health of the infants of Cleveland with the conviction that the mortality rate could be materially reduced. As a result of these circumstances the idea of a central dispensary for the care of sick infants and of prophylactic branches located in the different congested areas of the city for the protection of the well infants was conceived and brought to fruition.

In 1911 as the result of the generosity of Mr. and Mrs. J. H. Wade, friends and admirers of Dr. H. H. Powell, professor of obstetrics of Western Reserve University and a member of the Medical Board of the Babies' Dispensary and Hospital, the Babies' Dispensary and the Milk Laboratory were built on East Thirty-Fifth Street as a memorial to their mother, Mrs. Anna R. Wade. It was at that time, of course, understood that the hospital building would adjoin that of the new dispensary at a not far distant date. In the meantime, however, three unforeseen factors altered these plans. The first was the untimely and tragic death of Dr. Edward F. Cushing. The second was the crystallization of the idea to bring on one plot of ground a new medical school and the hospital units necessary for the teaching of the medical students in the departments of medicine, surgery, obstetrics, and pediatrics. The third unforeseen factor was the World War.

During these dark years there was for the Babies' Dispensary and Hospital one bright and encouraging light, namely, the generosity and understanding of Mr. H. M. Hanna who, particularly out of devotion to the memory of Dr. Edward F. Cushing, gave sufficient funds to the Board of Trustees of the Babies' Dispensary and Hospital to assure the ultimate erection of the hospital building. The tremendous increase in building costs which characterized the postwar years, however, required the collection of additional funds. These finally were obtained in 1923 when the public of Greater Cleveland responded in a magnificent and generous manner to a campaign appeal jointly made by the Babies' Dispensary and Hospital and the Maternity Hospital. The process of erecting the building was immediately begun and the Babies and Childrens Hospital was opened as a memorial to Dr. Edward F. Cushing on December 5, 1925, nearly nineteen years after the original plan had been conceived. The sixth floor, devoted entirely to research, was dedicated to the memory of Mr. H. M. Hanna, the Hospital's greatest benefactor. The seventh floor ward, a fresh-air ward, is a memorial to Dr. John H. Lowman; the

second floor, the contagious division, to Anna R. Wade and Ellen Garretson Wade, mother and wife of Mr. J. H. Wade; and many of the private and semiprivate rooms, individual cubicles, and even some of the laboratories are memorials to friends and relatives of generous benefactors.

HOSPITAL FACILITIES

The Babies and Childrens Hospital building is seven floors high, with a basement and a subbasement. In the subbasement are located the sterilization room for mattresses and a laundry collecting room. In the basement are the milk laboratory and diet kitchen, the pharmacy, the autopsy room (which is no longer used since the opening of the Institute of Pathology), the amphitheater, lecture and rest rooms

The Babies and Childrens Hospital of Cleveland

and lockers for nurses, and the admitting suite, with a special entrance, for patients ill with contagious diseases.

The first floor is occupied by the lobby, the admitting suite for ward patients with its adjoining clinical laboratory, a general ward with its bathroom, utility room, and diet kitchen, by offices for the departments of administration and nursing, by consultation offices for the full-time staff, by the upper half of the amphitheater, and by the entrance corridor and toilet facilities for the medical students coming to the amphitheater for lectures or clinics.

The second floor is specially arranged for the care of patients ill with contagious diseases and is the first contagious disease division to be located in a noncontagious institution. A special elevator, running only to this floor, brings the patients from the admitting suite in the basement.

The third floor provides rooms for private and semiprivate patients and a ward for older children; and the fourth floor, two wards for babies and special suites for premature infants and for wet nurses.

The fifth floor has a room equipped for dental work, a small operating room for special work on newborn infants, six individual rooms, and two wards, one of which was planned for the study and care of psychopathic children.

The sixth and seventh floors were enlarged during 1929-1930 and at the present time offer the following services. The sixth floor is used for central and research laboratories, for offices of the full-time men, for hospital record rooms, and for the library which is supported by an endowment fund of $60,000 set aside for this particular purpose out of the income from S.M.A. On the seventh floor are located a photographic studio for the taking of still and moving pictures and for other photographic work, such as the making of lantern slides; a roentgenographic suite of two rooms with an adjoining viewing room; living quarters for the residents and assistant residents; and a ward with southern exposure for the care of chronically ill children. The uncovered part of the roof is available for heliotherapy.

The wards on each floor are located at the easterly and westerly ends of the building and are as long as the building is wide from north to south. As a result, each ward has windows on three sides and is well lighted and ventilated. All of the utility rooms are located on the north side of the building and all of the private rooms and special suites on the south. Two small porches are located on each floor adjoining the wards on the south side of the building. On the north side are large screened porches, twelve feet wide, for use during the heat of the summer. The wards, utility rooms, and diet kitchens open on to the north porches and so transform these porches into open-air wards with facilities quite as convenient as those existing for the regular wards.

The temperature of the wards is thermostatically controlled and the degree of humidity can be regulated automatically during the colder months of the year.

The building is practically soundproof, the ceilings of the corridors, individual rooms, utility rooms, and diet kitchens being padded with felt and those of the wards being made of acoustic plaster. The corridor floors and the walking paths in the wards are covered with rubber. This combination of rubber floors and soundproofed ceilings has made this hospital much more quiet than children's hospitals ordinarily are. This is undoubtedly of benefit to patients, nurses, and physicians.

Each ward is divided into twelve cubicles, and each cubicle is arranged to care for one bed. In the wall of each cubicle are electric connections for therapeutic lamps, electric fans, and other therapeutic apparatus, such as hot plates and steam kettles. Facilities for washing hands are liberally provided in order to encourage the frequent application of this protective procedure.

The number of beds available at the Babies and Childrens Hospital is as follows: ward beds, 110, including 24 for contagious diseases; private beds, 16, including 5 for contagious diseases; and semiprivate beds, 11; making a total of 137. During the five years from 1927 to 1931, inclusive, the occupancy rate for ward beds averaged 92 per cent.

The medical care of the ward patients is supervised by the staff of the department of pediatrics, while the private rooms, including those on the contagious division floor, may be used for patients by any reputable physician in the city of Cleveland. The Hospital is a "pediatric clinic," that is, a clinic for "medically ill" babies and children; only such "surgically ill" infants are cared for as need very special pediatric attention. "Surgically ill" children are cared for by the department of surgery in Lakeside Hospital, with consultation service from the department of pediatrics with regard to feeding and supervision.

OUT-PATIENT DEPARTMENT

The out-patient department of the Babies and Childrens Hospital is the pediatric division of the out-patient department of University Hospitals. This division is composed of a general pediatric clinic, nine special clinics, and a welfare clinic for infants and children. The general clinic serves as a differentiation clinic for all children coming to the out-patient department of the University Hospitals. In the pediatric clinic there are an admitting room with desks for the pediatric resident in charge of admissions, for the pediatric nurse, and for the pediatric social service worker, a waiting room, a weighing and measuring room, an isolation suite of six cubicles with an outside exit, a spacious, combined clinical laboratory and library, and a dental room. The total number of visits was 23,786 for the year 1932 and 23,744 for the year 1933.

The special clinics are listed as follows: allergy, cardiac, dental (preschool), diabetic, endocrine, enuresis, goiter, mental health, and syphilis.

The welfare clinic for infants and children directly adjoins the pediatric clinic. It consists of a waiting room, a weighing and measuring room, two examining rooms, and an office. These rooms during the afternoons are used by the Children's Bureau of Cleveland, which, in cooperation with the Humane Society and the County Welfare Board, employs members of the department of pediatrics for the examination and supervision of their charges. On two forenoons the Bureau of Child Hygiene of the Division of Health of the City of Cleveland uses these rooms for infant welfare work. Conferences with parents of behavior-problem children are also held in these quarters on three forenoons. The use of these rooms by these various agencies enables the members of the house staff on service in the out-patient department to come into contact with this type of pediatric work.

Four extramural dispensaries, of which three are infant welfare stations of the Bureau of Child Hygiene, are also staffed for pediatric teaching of clinical clerks by members of the department of pediatrics. The fourth extramural dispensary, formerly known as the Babies' Dispensary, is under the supervision of the Cleveland City Hospital as already described.

STAFF OF THE HOSPITAL AND OUT-PATIENT DEPARTMENT

The visiting staff of the Babies and Childrens Hospital and of the pediatric clinic of the out-patient department of the University Hospitals is at present composed of the director of pediatrics and twenty-two associates and assistants. The house staff is made up of two residents who alternate on a three-month service in the Hospital and in the out-patient department and are in charge of admissions; four assistant residents who rotate on a six-month service at the Babies and Childrens Hospital and at the Cleveland City Hospital, each spending at the Babies and Childrens Hospital three months in the Hospital and three months in the out-patient department and at the City Hospital three months in the department of contagious diseases and three months in the department of pediatrics; and five internes. Two internes are on a straight pediatric service for one year and three are on a two-year rotating service majoring in pediatrics, taking four months each as minor services on medicine and surgery. The department of pediatrics also maintains an assistant resident on its service at the Rainbow Hospital for Crippled and Convalescent Children. Two workers from the social service department are assigned to pediatric cases at the Babies and Childrens Hospital and at the Rainbow Hospital.

A thorough training in pediatrics is offered to physicians who are accepted as internes at the Babies and Childrens Hospital in the form of a continuous course covering four or six years. Physicians on this service will thus have the opportunity to plan ahead for that period of time. Such a course includes, beyond the interne-

ship, the following successive positions: (a) four assistant residencies, duration one year, with service as described above; (b) four residencies, duration one year, two being available in the Babies and Childrens Hospital as described above, and two in the Cleveland City Hospital, one in the department of contagious diseases and one in the department of pediatrics; and (c) two teaching and research fellowships at the Babies and Childrens Hospital, one for the duration of one year and one for two years. Completion of such a training will make a physician eligible for admission to the American Academy of Pediatrics and for examination to be certified as pediatrician by the American Board of Pediatrics.

THE TEACHING STAFF AND CURRICULUM

The present teaching curriculum in the department of pediatrics provides in the third year a course of thirty-two lectures giving the fundamentals of diseases of infants and children, including contagious diseases and mental hygiene. Instruction in the fourth year is of two kinds: (1) amphitheater clinics and (2) clinical clerkships. The latter course includes two hundred and twenty-four hours of bedside training and ninety-six hours of dispensary service; the amphitheater clinics, numbering thirty-two hours, are presentations to the entire class of cases typical of the general field of pediatrics. At the present time these thirty-two hours are being used to give sixteen clinics of two hours each on alternate weeks. At these clinics a thesis is presented by one of the members of the Senior class.

The clinical clerkship and dispensary instruction is given to sections of one-tenth of the class for a period of eight weeks, halves of each section alternating on "inside" and "outside" services. The four weeks on "inside" service are spent as follows: (a) on the wards at the Babies and Childrens Hospital where practical experience is had daily in the taking of histories, making physical examinations and doing routine laboratory work and in follow-up of the patients' progress—all under the supervision of members of the visiting staff of the department of pediatrics; (b) at the pediatric clinic of the out-patient department of the University Hospitals, including conferences in mental hygiene, five afternoons a week; and (c) in group meetings and daily ward rounds with members of the visiting staff, where practical demonstration is given in problems concerning the diagnosis, care, and treatment of diseases of infants and children.

The four weeks on the "outside" service are spent as follows: (a) in demonstrations and conferences in the departments of contagious diseases and of pediatrics at the Cleveland City Hospital, two mornings a week; (b) in dispensary service at the East Thirty-Fifth Street Dispensary of the City Hospital, five afternoons a week; (c) in preventive pediatric work at infant welfare stations of the Bureau of Child Hygiene of the Division of Health, two mornings a week; (d) at the Cleveland Child Guidance Clinic where instruction is given in mental hygiene, one afternoon a week, and at the Nursery School of Western Reserve University, one morning during the four-week period; and (e) in the preparation of theses on assigned pediatric subjects.

Members of both the full-time and the part-time visiting staff take part in the teaching of students in the third and fourth years.

RESEARCH

Research work at the Babies and Childrens Hospital has been principally in the direction of fundamental studies in growth and nutritional and metabolic disorders, while at the Cleveland City Hospital infectious diseases principally have been studied in the department of contagious diseases and pharmacologic problems in the department of pediatrics.

It is in general the policy of the Babies and Childrens Hospital to encourage the younger men to get first a very thorough and fundamentally sound clinical training and experience in the diagnosis, treatment, and prognosis of diseases of infants and children and, in particular, to use all of the known clinical and laboratory methods to obtain the maximum amount of information regarding the individual case before embarking upon individual research problems. The younger men, during the fellowship period, are given the opportunity to work in conjunction with the older men in the department. The statements just made do not, however, imply that individual research on the part of the younger men is actively discouraged. It simply is the conviction of the head of the department that the investigative efforts by the younger men are more valuable to themselves and to the science of pediatrics if the men are associated during the apprenticeship years with investigators of experience.

Another conviction of the head of the department is that a rather inclusive library in the form of textbooks, monographs, and current journals is one of the greatest, if not the greatest, asset to young men who are interested in broadening their knowledge and in deepening their thinking. It is for this reason that the first endowment fund which he created was the one to insure an adequate income for the maintenance of an excellent library.

A great deal of practical effort of an investigative sort that is of value to the individual physician, to the institution, and, of course, also to the patient from the standpoint of diagnosis, teaching and records has been intensively fostered at the Babies and Childrens Hospital: namely, roentgenographic study of pathology in the patients by members of the pediatric staff in charge of the individual patients with the aid of competent technical assistance and photographic registration on the still and moving film of clinical pictures. In this manner a valuable library of roentgenograms, still pictures, and moving pictures is being collected which, when a certain stage of development will have been reached, will be of great value in undergraduate, and particularly in postgraduate, teaching. For the present the responsibility for postgraduate instruction has not been accepted.

Critical Review

NEUROLOGIC CONDITIONS IN INFANTS AND CHILDREN

R. Cannon Eley, M.D.
Boston, Mass.

ENCEPHALITIS

SINCE von Economo's[1] description of "encephalitis lethargica" in 1917 various forms and manifestations of encephalitis have been encountered and described.

The syndrome has been observed as occurring not only as the primary or idiopathic form, i.e., as the result of an invasion of the central nervous system by a virus, but also secondary to acute infections,[2, 3] common contagious diseases,[4-14] chronic lead poisoning,[15-20] postvaccinial,[21-25] and many other conditions. The idiopathic form of the disease, as originally described, was characterized by asthenia, cranial nerve palsies, and lethargy. However, it soon became recognized that the manifestations were so protean, and varied so markedly in both their severity and in their intensity that the term "encephalitis lethargica" was not applicable and for this reason it was soon replaced by the term "epidemic encephalitis." Following this, attempts were made and are still being made, to divide the disease into elaborate classifications, but they have not been successful, for frequently it is impossible to place a case in any one group or type, as a single case may pass through various types in the course of the disease. However, the employment of such broad terms as "meningoencephalitic type" when the meningeal symptoms predominate and "encephalomyelitic type" when the cord symptoms predominate not only seems advisable but indicated, as the terms immediately connote not only the symptomatology but also the site of the pathology.

In the course of time, and with the disease becoming more prevalent, the epidemiologic features and the varied clinical manifestations of epidemic or idiopathic encephalitis became better recognized. Thus different waves of epidemic encephalitis, like different epidemics of poliomyelitis, present definite outstanding groups of symptoms for that epidemic. Age and seasonal features became recognized, and, although the disease does occur at any period of life,[26] it is more frequently encountered in that period between ten and twenty years of age than at any other time; epidemics are more commonly observed in the winter than in the summer months, yet the occurrence of epidemics in the summer is not rare. Furthermore, the clinical manifestations of the so-called "summer" epidemic encephalitis differ quite markedly from that observed

From the Infants' and Children's Hospitals and the Department of Pediatrics, Harvard Medical School, Boston, Massachusetts.

during the winter months. As a result of this difference the summer forms of the condition are spoken of as Type A and the winter forms as Type B epidemic encephalitis. The form occurring in the summer is characterized by a more acute onset, more marked meningeal involvement, a greater tendency to delirium or coma, and more pronounced muscular rigidity than is that form occurring in the winter. Paralyses of the ocular muscles are common in Type A but rare in Type B, while paralyses of the muscles of the extremities and head are seen more frequently in Type B than in Type A. Sequelae, such as changes in personality, reversal of sleep syndrome, or the development of Parkinson's syndrome are much more frequently encountered following Type B than Type A. However, in different epidemics, this seasonal distribution of the two types may not be present, or there may be no sharp line of demarcation between them—one gradually assuming the characteristics of the other. The recent outbreak at St. Louis in August and September of 1933 serves as another excellent example of the variability of this disease.

During this epidemic there were many divergencies from what is commonly considered the usual form of the disease. The symptomatology made it compare very closely to that seen during the epidemic in Japan in 1924.[27] Ptosis, ophthalmoplegia, and strabismus were rarely encountered, whereas involvement of the central nervous system was an outstanding feature as is clearly demonstrated in the following statement: "Demonstrable meningeal involvement is practically uniform in the recognizable cases."[28] In the majority of cases the first manifestation was the sudden onset of meningeal irritation which was then followed by rapid development of stupor, apathetic facies, stiff neck, and rigidity of the spine; muscular tremors were common, and there was a marked tendency for the muscles to become very rigid. In many instances the entire course of the disease was relatively short when compared with the summer type of epidemic encephalitis. This is clearly demonstrated by the fact that as the temperature declined, usually after three or four days, there was a gradual disappearance of the symptoms. Another striking difference was the age group as the disease was more prevalent among the older age groups than in the younger age groups. As with the other types of encephalitis, there was no racial or sex predilection. Postmortem examinations showed that the lesions in the central nervous system were similar to those observed in other epidemics resulting from a filtrable virus and differ only so far as the lesions are much more diffuse and show less tendency to involve the basal ganglia and brain stem.

As the result of the intense study of the epidemic occurring at St. Louis, many valuable and important contributions have been forthcoming. Due to the failure to demonstrate any cultivable organism either from the blood or cerebrospinal fluid, it was early suggested that the causative agent was probably a filtrable virus. Experimental studies with susceptible white mice soon confirmed this supposition. Webster and Fite[36] in an effort to determine the nature of the virus performed a series of experiments and showed that the serums obtained from convalescent patients in St. Louis possessed definite and specific protective bodies against this type of encephalitis alone and that serums obtained from normal individuals did not possess these specific bodies. Neal[29] suggested it was more logical to assume that there had been a common

infecting agent which had attacked the central nervous system in different anatomical sites and thereby had given rise to varied clinical syndromes rather than to assume that there had been several different infecting agents all of which had given rise to similar changes in the central nervous system, but with different clinical manifestations. However, if the former hypothesis were true, would not one expect that the serums of the individual patients would possess a common neutralizing power? In fact it is on this very point, namely the specificity of the neutralization tests, that Rivers and Schwentker[31] conclude that the virus of louping-ill may produce an encephalitis in man which clinically can hardly be distinguished from that which occurred at St. Louis.

In view of the recently reported clinical manifestations of louping-ill in man and the fact that the symptomatology and clinical course of this disease closely simulates the symptomatology observed during the epidemic of encephalitis in St. Louis in 1933, a brief consideration of louping-ill in man and the recent epidemic of encephalitis is interesting.

A review of the literature fails to reveal any individual from whom the virus of louping-ill has been recovered, or any record of the occurrence of this disease in man, yet three individuals working with this virus[30] in the hospital of the Rockefeller Institute for Medical Research in New York City presented such clinical and serologic evidence that a diagnosis of the disease was warranted.[31] In all three patients general malaise, headache, and a moderate febrile reaction preceded the appearance of diplopia, blurred vision, nausea, vomiting, stiff neck, and drowsiness. The cerebrospinal fluid, examined on several occasions in each case, presented chemical and cytologic changes compatible with those encountered in encephalitis. In two instances an initial diagnosis of tuberculous meningitis was made but as no organisms could be cultured or demonstrated in the withdrawn cerebrospinal fluid, and as both patients recovered, a final diagnosis of encephalitis was made. In the third patient an initial diagnosis of encephalitis was made at the onset of the illness. As encephalitis was the final diagnosis in all three instances, efforts were instituted in two of the patients to determine the nature of the virus responsible for the disease. Unfortunately the workers could not recover the virus in either the blood or the cerebrospinal fluid of either patient. However, by means of neutralizing tests[32] they were able to demonstrate the presence of specific antibodies in the serum of these individuals and to demonstrate their absence in individuals who had not had the disease and who had not been exposed to the virus. In view of this observation, the attending circumstances under which all three patients became ill and the fact that the virus cannot always be recovered from animals who are known to be suffering from the disease, there can be little doubt that these individuals contracted louping-ill while performing experimental work with the specific virus.

When louping-ill is experimentally produced in mice, there follows an incubation period of from six to seven days before the animals develop any signs or symptoms referable to the central nervous system. However, with the development of signs of meningeal irritation the mice usually die within two or three days, and at autopsy lesions typical of those seen in encephalitis can be demonstrated.[33, 34] Usually the virus does not appear in the blood stream until the second day after the intranasal inoculation, but from that time until the animals become sick it is practically always present. With the development of cortical lesions

and coincident with the apparence of signs of meningeal irritation, the virus disappears from the blood stream.[35] In retrospect one can readily see this course manifested by the three individuals who were working with the virus, and at the same time an adequate explanation is offered for the negative results that were obtained when efforts were made to recover the virus from the blood stream and the cerebrospinal fluid of the patients as this effort was not instituted until *after* signs of encephalitis had developed.

As the symptomatology presented by these three patients was similar to that observed during the epidemic of encephalitis at St. Louis, it was naturally suggested that the virus of louping-ill might be the etiologic agent involved in this epidemic. Accordingly efforts were made to prove or disprove this relationship. Serums obtained from the louping-ill patients did not neutralize[31] the virus obtained from the St. Louis patients, nor did serums which would protect mice against one hundred lethal doses of louping-ill virus have any effect upon mice intranasally inoculated with the virus obtained from the St. Louis patients.[36] Therefore, one is forced to conclude that the recent epidemic was unrelated to that form of encephalitis which has been observed in man for the first time.

It would be of epidemiologic as well as of etiologic importance to know whether or not ground brain tissues obtained from the St. Louis patients would produce louping-ill when injected intracerebrally into sheep. Yet when one realizes that the neutralization tests for the virus of herpes, rabies, and poliomyelitis are all specific in their reactions, it does not seem probable, even though the clinical manifestations may be the same, that the various types of epidemic encephalitis are due to a common etiologic agent unless there are several strains of the same virus. However, from the literature one is left with the impression that the St. Louis epidemic was simply an unusual and rarely encountered form of primary or idiopathic encephalitis, rather than an encephalitis due to a new and hitherto unrecognized virus.

ACRODYNIA

Although this interesting syndrome was first described in 1903,[37] it did not gain prominence until 1914 at which time a series of cases were reported by Swift[38] in Australia. Since then the literature has gained voluminous proportions and numerous names have been applied to it, such as Swift's disease, pink disease, Feer's disease, erythredema, vegetative neurosis, acrodynia, etc.

The various factors that have at one time or another been considered as of etiologic importance are as numerous as the terms applied to the disorder. In 1828 there occurred an epidemic of a disease in Paris[39] clinical manifestations of which closely simulated those now described as occurring with acrodynia yet differing from it in its age distribution; i.e., only adults were affected. Subsequently it was decided that the disturbance had been due to arsenic. As the symptomatology of the two conditions was so similar, it was naturally suggested that acrodynia was the result of arsenic poisoning. There are a few today who still support this view.[40] Ergotism[41] and excessive exposure to sunlight[42] have been considered as etiologic possibilities. Among the various factors that have been considered, the two receiving the greatest support and credence are infection[43, 44] and dietary deficiency.[45, 46, 47] Of the

infections that might precipitate this syndrome, influenza has received the greatest attention. This can perhaps be explained by the fact that acrodynia had not been definitely and universally recognized as a clinical entity until between 1914 and 1920, and it was during this period of time that the country was recovering from a widespread epidemic of influenza. The occurrence of acrodynia in certain localities and its absence in others has been considered as of epidemiologic significance, i.e., it might be of an endemic infectious nature. However, the extreme rarity of the disease in more than one member of the family, and the absence of marked variations in the yearly incidence of the disease do not lend support to this view. Although infections can be demonstrated in practically every patient suffering from acrodynia, it is usually of a secondary nature. The similarity of some of the clinical manifestations to pellagra has already been mentioned yet the inability to reproduce experimentally the disease by the administration of deficiency diets and likewise the inability to clinically relieve the patients by the administration of selective diets certainly make one hesitate to accept this explanation.

As the symptomatology of this clinical syndrome has been described so many times, only those symptoms referable to possible disturbances within the central nervous system will be considered. "When the presenting symptoms, the physical signs, and laboratory data are considered carefully, it is apparent that there is present a widespread and fundamental derangement involving many organs and tissues of the body. Therefore, it does not seem unreasonable, whatever the precipitating cause, to relate certain of the outstanding features of the disease to disturbance of the central nervous system together with a dysfunction of the autonomic nervous system. The latter suggested first by Feer."[48]

If one considers carefully the varied manifestations of acrodynia, it will become apparent that a large number of the symptoms are referable to changes within the nervous system. Thus apathy, coma, convulsions, paralysis, muscular weakness, hyperesthesia, and hypotonia can be attributed to involvement of the brain and spinal cord. That evidence of involvement of the autonomic system is not wanting is clearly shown by marked vasomotor disturbances in the hands and feet, sweating, tachycardia, hypertension, elevation of blood sugar, increased metabolic rate, dilatation of the pupils and photophobia, alopecia, salivation, rhinorrhea, etc. In fact, it would appear that the chief disturbance is one involving the entire nervous system in a very diffuse and widespread manner thereby producing changes which involve the entire body.

If this conception is true, one would expect to find pathologic disturbances involving either the sympathetic chains, or a central lesion involving particularly the centers of autonomic control. As the termination of the disease is usually favorable, pathologic studies have of necessity been somewhat limited. However, such studies that have been completed have shown degenerative peripheral demyelinization, and cellular infiltration of the cord and nerve roots. Paterson and Greenfield[49] have described cellular infiltration in the spinal cord with demyelinization of the peripheral nerves; Kernohan and Kennedy[50] have not only noted demyelinization but also degeneration of the cord extending as high as the base of the brain. Wyllie and Stern[51] confirmed the observations of Paterson and Greenfield and noted that there was a marked chromatolysis of the central type which involved the anterior horn cells;

Orton and Bender[52] demonstrated chronic lesions in the lateral horns in the lumbar and thoracic region; they were unable to demonstrate the presence of those nerve cells that are considered to serve as the connecting link between the sympathetic nervous system and the spinal cord. In two cases of acrodynia that were very carefully studied, Warthin[53] found extreme edema and slight meningeal irritation of the central nervous system.

The pathologic changes that have been described by the above authors would appear to be of significance, but until the underlying etiologic agent has been determined, extreme caution should be exercised in accepting them in their entirety. This is especially true when one realizes that those patients who die are usually in a very poor, if not critical, nutritional state, have usually been ill for a relatively long period of time, and that the cause of death is some intercurrent or secondary infection which in such a debilitated individual might be capable of producing widespread "toxic" influences. An excellent discussion of this phase of the disease may be found in the recent paper by Blackfan and McKhann.[48]

Until the etiology of acrodynia is known, treatment must of necessity be simply directed along symptomatic channels. Various endocrine preparations[54, 55] have been tried without avail; the removal of tonsils and adenoids has been suggested by some;[44] vaccines have also been employed without remarkable effect.[56] As the disease tends to run a chronic course, efforts directed toward the maintenace of a good nutritional state, plus such local measures that will afford comfort to the patient, appear to offer the most satisfactory form of treatment at the present moment.

As the patient improves, there is a noticeable change not only in the mental state and general activity but in the reaction of the patient to his environment. As recovery may be slow and protracted and as exacerbations may occur, it is not advisable to urge the child in either strenuous mental or physical activity.

PROGRESSIVE PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY

In view of the number of case reports in the literature concerning beneficial symptomatic treatment, a brief review of this condition is of interest.

Although Duchenne[57] is usually considered as having published the first description of this disease in 1849, he was actually antedated by Semmla in 1834 and Costa and Gioga in 1836. During the following twenty years, the primary myopathies were gradually differentiated from the atrophies of nervous origin. The former group has been further subdivided according to the anatomic distribution of involvement and at the present time five types of this disease are recognized:

1. The Duchenne pseudohypertrophic, and most common form
2. The pseudohypertrophic form occurring early in the muscles of the shoulder
3. A form quite similar to the Duchenne type but not accompanied by pseudohypertrophy and known as the Leyden-Moebius type
4. The facioscapulohumeral type of Landouzy-Déjérine
5. Juvenile form of Erb. This type is quite similar to the facioscapulohumeral form but without the typical so-called "myopathic facies."

The etiology of this condition continues to be poorly understood, and,

as one might expect, numerous factors have at one time or another been considered. Goldstein[58] has suggested that it might be the result of some endocrine disorder with particular reference to the pineal body; Barker[59] has demonstrated definite physical and metabolic evidences of endocrine dysfunction; Landé[60] has reported a case associated with dystrophia adiposogenitalis; Janney, Goodhart and Isaacson[61] have found changes indicative of general endocrinologic disturbances such as dry brittle hair, pigmentation of the skin, acromegalic features, trophic changes in the nails, and unusual distribution of subcutaneous fat. Kuré and Okinaka[62] have concluded that the disease is the result of an alteration in the autonomic innervation of the muscles and on the basis of their experimental work have devised their form of treatment. Syphilis, as well as other diseases of the central nervous system, has been found associated with this disease, but there is no evidence to support the view that the condition is the result of any of these disturbances. Heredity unquestionably plays an important part, and it is not unusual to see two or more members of the same family affected. This is particularly true in regard to brothers. A recent study of a family in a secluded Swiss village where accurate records for three hundred years were available has shown that the hereditary features, according to the mendelian theory, are of the recessive mode.

Studies in blood chemistry have largely concerned themselves with creatine and glycogen metabolism, as abnormalities in both of these substances have been repeatedly demonstrated. McCrudden and Sargent[63] pointed out the low blood sugar content present in their patient and attempted to explain the muscular weakness by the low glycogen content of the muscles. Milhorat, Techner and Thomas[64] have shown that these patients have a creatinuria even on a creatine-free diet and excrete creatine almost quantitatively. This is a complete reversal from that which occurs in the normal individual. Similar observations have been made by Levine and Kristeller.[67]

Clinically the disease is not difficult to recognize as the pseudohypertrophy of the muscles, particularly the muscles of the calf and thigh, and the pronounced muscular weakness are outstanding features. As a rule the first indications of the disease are detected by the parents who observe that the child is not walking as early as he should be, or if he is walking, that his gait is uncertain and awkward. These symptoms become progressively worse so that the child becomes unable to arise from the floor and finally becomes a complete invalid. It is worthy of note that frequently confinement to the bed on account of some illness may hasten the time of complete helplessness. Occasionally it may be necessary to differentiate this disease from other conditions, particularly those of neural origin. However, the age of onset, the hereditary tendency, the site of involvement, and the absence of fibrillary twitchings and spasticity will readily separate the true primary myopathies from those occurring secondary to changes within the central nervous system.

Treatment in the past has been both discouraging and futile although occasionally improvement has been observed following the administration of a high carbohydrate diet or the use of polyglandular extracts. Recently encouraging reports have appeared in the literature and at the present time two methods of therapy have been offered.

Based on their experimental evidence Kuré and Okinaka[62] in 1930 suggested that those patients should be treated with pilocarpine and

adrenalin and have offered the following procedures: From 0.2 to 0.3 c.c. of 1 per cent adrenalin solution and from 0.1 to 0.2 c.c. of 1 per cent solution of pilocarpine hydrochloride are injected subcutaneously every day or every other day until from fifty to sixty doses have been administered without interruption. The authors treated twelve of their patients in this manner and were able to obtain improvement in six, "functionally" complete cure in two, but no improvement in the others which, however, were far advanced cases. The first evidence of improvement usually appeared about the time of the fiftieth injection and persisted for several months after the injections had been discontinued. Except for slight palpitation in a few cases, no unfavorable results were noted. Hough,[68] in 1931, treated sixteen patients with this method and obtained improvement in every case although several of them were quite advanced. Of this group eight showed slight improvement; four moderate improvement; and four showed marked improvement; in one case that was comparatively early "a symptomatic and functional recovery" was obtained. Both authors are careful to point out that this form of therapy is *not* a cure, that relief is only temporary, and that the treatments have to be repeated at regular intervals. In a second communication in 1933 Hough[69] states that the procedure "is of sufficient value to justify its continued use in this disease."

The second therapeutic procedure was offered by Milhorat, Techner and Thomas[64] in 1932 and is based on the effect of glycine administration upon creatine metabolism. These authors treated three patients suffering from progressive pseudohypertrophic muscular dystrophy by the oral administration of 5 gm. of glycine daily for eight periods of three months each with an interval of three weeks between each period of treatment. Chemical studies showed that the creatinuria was definitely increased and that after a period of a few weeks this gradually decreased until it had reached its former level, although the glycine had not been discontinued. Clinically there was a slow disappearance of fatigue which was accompanied by increased activity and an improvement in motor acts that were formerly executed in a very poor and awkward manner. Chanutin, Butt, and Royster[70] treated five patients in this manner with improvement in three; the other two showed improvement at first but soon became worse. Mettle and Slocum[71] treated three patients with marked improvement in two and slight improvement in the other one. Beneficial therapeutic results have also been reported by Kostakow and Slauch.[72] Beard and Tripoli[73] obtained improvement in their patients by the oral administration of either glycine or glutamic acid and also observed a creatinuria, which after two or three weeks gradually disappeared. This, they considered, indicated a retention of muscle creatine in the body. Harris and Brand[74] were unable to detect any striking improvement as reported by Thomas, Milhorat and Techner.[75] In an attempt to evaluate the efficacy of the two procedures, i.e., the use of adrenalin and pilocarpine and the use of glycine or glutamic acid, Voshell[76] treated fourteen patients and concluded that the patients who received adrenalin and pilocarpine responded more favorably. Patients who were changed from this form of treatment to the glycine treatment showed definite signs of retardation of the process. The suggestion is made that the two procedures be combined, thereby furnishing muscle tissue per se with glycine and at the same time stimulation to the autonomic nervous system.

In conclusion it would appear that although beneficial results have not been obtained in all cases that have been treated, the evidence is such that would indicate the employment of one or both methods in the treatment of this progressive and disabling disease as long as there is no evidence to suggest that its employment is detrimental to the patient. Obviously, further studies are necessary before the methods can be fully accepted.

REFERENCES

1. Economo, C. von: Wien. klin. Wchnschr. **30**: 581, 1917.
2. Grinker, R. R., and Stone, T. T.: Arch. Neurol. & Psychiat. **20**: 244, 1928.
3. Low, A. A.: Arch. Neurol. & Psychiat. **23**: 697, 1930.
4. Husler, J., and Spatz, H.: Ztschr. f. Kinderh. **38**: 428, 1924.
5. Jochims, J.: Ztschr. f. Kinderh. **45**: 326, 1928.
6. Ford, F. R.: Am. J. Dis. Child. **37**: 1046, 1928.
7. Askin, J. A., and Zimmerman, H. M.: Am. J. Dis. Child. **38**: 97, 1929.
8. Singer, Ludwig: Ztschr. f. Kreislaufforsch. **20**: 338, 1928.
9. Gordon, M. H.: Experimental Production of the Meningoencephalitis of Mumps, Lancet **1**: 652, 1927.
10. Casparis, Horton R.: Cerebral Complications in Mumps, Am. J. Dis. Child. **18**: 187, 1919.
11. Zimmerman, H. M., and Yannet, Herman: Non-Suppurative Encephalomyelitis Accompanying Chicken-pox, Arch. Neurol. & Psychiat. **26**: 322, 1931.
12. Ford, F. R.: The Nervous Complications of Measles, Bull. Johns Hopkins Hosp. **43**: 140, 1928.
13. Greenfield, J. G.: Brain **52**: 171, 1929.
14. Ferraro, A., and Schaffer, I. H.: Arch. Neurol. & Psychiat. **25**: 748, 1931; Ibid. **27**: 1209, 1932.
15. McKhann, C. F.: Lead Poisoning in Children. The Cerebral Manifestations, Arch. Neurol. & Psychiat. **27**: 294, 1932.
16. Ruddock, J. C.: Lead Poisoning in Children with Special Reference to Pica, J. A. M. A. **82**: 1682, 1924.
17. Wilcox, H. B., and Caffey, J. P.: Lead Poisoning in Nursing Infants, J. A. M. A. **86**: 1514, 1926.
18. Park, E. A.; Jackson, Deborah; and Kajdi, Laslo: Am. J. Dis. Child. **41**: 485, 1931.
19. Vogt, Edward C.: Am. J. Roentgenol. **24**: 550, 1930; J. A. M. A. **98**: 125, 1932.
20. Caffey, John: Radiology **17**: 957, 1931.
21. Troup, A. G., and Hurst, E. W.: Disseminated Encephalomyelitis Following Small-Pox, Lancet **1**: 566, 1930.
22. McIntosh, J., and Scarff, R. W.: Reaction of the Central Nervous System to Vaccinia Virus, J. Path. & Bact. **33**: 483, 1930.
23. Eckstein, A.: Investigations on the Cause of Vaccination Encephalitis, Arch. Dis. Childhood **7**: 105, 1932.
24. Jorge, Recordo: Post-Vaccinal Encephalitis, Lancet **2**: 215, 1932; Lancet **2**: 267, 1932.
25. Thompson, Richard: The Etiology of Post-Vaccinal Encephalomyelitis, Arch. Path. **12**: 601, 1931.
26. Report of Matheson Commission, "Epidemic Encephalitis," New York, 1929, Columbia University Press.
27. Kaneko, R.: Jap. Med. World **5**: 237, 1925.
28. Leake, J. P.: Encephalitis in St. Louis, J. A. M. A. **101**: 928, 1933.
29. Neal, Josephine: Types of Epidemic Encephalitis. A Comparison of the Cases Seen in St. Louis in 1933 With Those Seen in New York City, Am. J. Pub. Health **23**: 1144, 1933.
30. Greig, J. R.; Brownlee, A.; Wilson, D. R.; and Gordon, W. S.: Vet. Rec. **11**: 325, 1931.
31. Rivers, Thomas M., and Schwentker, Francis F.: Louping Ill in Man, J. Exper. Med. **59**: 669, 1934.
32. Schwentker, F. F.; Rivers, T. M.; and Finkelstein, M. H.: J. Exper. Med. **57**: 955, 1933.
33. Webster, L. T., and Fite, G. L.: Proc. Soc. Exper. Biol. & Med. **30**: 656, 1933.

34. Elford, W. J., and Galloway, I. A.: J. Path. & Bact. 37: 385, 1933.
35. Fite, G. L., and Webster, L. T.: Distribution of Virus of Louping Ill in Blood and Brain of Intranasally Infected Mice, Proc. Soc. Exper. Biol. & Med. 31: 695, 1934.
36. Webster, L. T., and Fite, George: A Virus Encountered in the Study of Material from Cases of Encephalitis in the St. Louis and Kansas City Epidemic of 1933, Science 78: 463, 1933.
37. Selter, O.: Über Trophodermatoneurose, Arch. f. Kinderh. 37: 468, 1903.
38. Swift, W.: Erythroderma, Australian Med. Congress. Child. Dis. Sect., Lancet, 1914.
39. Chardon: De l'épidemie d'Acrodynie qui a rége a Paris et aux environs en 1828, Journal de Clinique, Hotel Dieu, 1830.
40. Meyer, F., and Weise, E. C.: Acrodynia; Arsenic as the Etiologic Factor, J. Pediat. 2: 750, 1933.
41. Debré, R., and Petot, C.: Presse méd. 35: 753, 1927.
42. Braithwaite, J. V.: On the Etiology and Treatment of Pink Disease, Arch. Dis. Child. 8: 1, 1933.
43. Byfield, A. H.: A Polyneuritic Syndrome Resembling Pellagra; Acrodynia Seen in Very Young Children, Am. J. Dis. Child. 20: 347, 1920.
44. Rodda, F. C.: Acrodynia; A Clinical Study of Seventeen Cases, Am. J. Dis. Child. 30: 224, 1925.
45. McClendon, S. J.: Yeast and Irradiated Ergosterol in Treatment of Acrodynia, J. A. M. A. 93: 455, 1929.
46. Weston, W., Jr.: Etiology of Acrodynia, South. M. J. 24: 378, 1931.
47. Zahorsky, J.: Pink Disease Treated with Yeast, Am. J. Dis. Child. 20: 347, 1920.
48. Blackfan, K. D., and McKhann, C. F.: Acrodynia; a Note on the Pathologic Physiology, J. Pediat. 3: 45, 1933.
49. Paterson, D., and Greenfield, J. G.: Quart. J. Med. 17: 6, 1923.
50. Kernohan, J. W., and Kennedy, R. L. J.: Am. J. Dis. Child. 36: 341, 1928.
51. Wyllie, W. G., and Stern, R. O.: Am. Dis. Child. 6: 137, 1931.
52. Orton, J. T., and Bender, L.: Bull. Neurol. Inst., New York 1: 506, 1931.
53. Warthin, A. S.: Arch. Path. & Lab. Med. 1: 64, 1926.
54. Sweet, G. B.: Treatment of Erythroedema, M. J. Australia (Supp.) 16: 510, 1927.
55. Swift, W.: Erythroedema or "Pink Disease," M. J. Australia 1: 155, 1921.
56. Vipond, A. E.: Further Observations Upon Acrodynia, Brit. J. Child. Dis. 23: 127, 1926.
57. Duchenne: Étude Comparée des Lèsions Anatomiques dans l'Atrophie Musculaire Progressive et dans la Paralysie générale, L'Union Medicale VII 51: 202, 1853.
58. Goldstein, H.: Muscular Dystrophy: "Pseudohypertrophic Type," Arch. Ped. 47: 377, 1930.
59. Barker, L. F.: Discussion of Five Patients Exhibiting Certain Forms of Muscular Wasting, M. Clin. North America 14: 109, 1930.
60. Landé, L.: Infantile Dystrophia Musculorum Progressiva Combined With Dystrophia Adiposogenitalis, Ztschr. f. Kinderh. 42: 355, 1926.
61. Janney, N. W.; Goodhart, S. P.; and Isaacson, V. I.: The Endocrine Origin of Muscular Dystrophy, Trans. Sect. Nerv. and Ment. Dis. Am. Med. Assn., p. 259, 1929.
62. Kuré, K., and Okinaka, S.: Treatment of Progressive Dystrophy by Combined Injections of Epinephrine and Pilocarpine, Klin. Wchnschr. 9: 1168, 1930.
63. McCrudden, R. H., and Sargent, C. S.: Hypoglycemia and Progressive Muscular Dystrophy, Arch. Int. Med. 17: 464, 1916.
64. Milhorat, A. T.; Techner, F.; and Thomas, K.: Significance of Creatine in Progressive Muscular Dystrophy and Treatment of This Disease With Glycine, Proc. Soc. Exper. Biol. & Med. 29: 609, 1932.
65. Magee, M. C.: Creatine and Creatinine Metabolism in Progressive Muscular Dystrophy, Am. J. Dis. Child. 43: 19, 1932.
66. Magee, M. C.: Excretion of Creatine and Creatinine in Progressive Muscular Dystrophy, Am. J. Dis. Child. 43: 322, 1932.
67. Levine, P. A., and Kristeller, L.: Am. J. Physiol. 24: 45, 1929.
68. Hough, G. DeN., Jr.: Progressive Pseudohypertrophic Muscular Dystrophy, J. Bone & Joint Surg. 13: 825, 1931.
69. Hough, G. DeN., Jr.: Metabolic and Therapeutic Studies in the Myopathies, J. A. M. A. 101: 1047, 1933.

70. Chanutin, A.; Butt, H. R.; and Royster, L. T.: A Study of Progressive Pseudohypertrophic Muscular Dystrophy in Children After the Administration of Glycine and Creatine, J. Biol. Chem. **100**: xxvi, 1933.
71. Mettle, H. B., and Siocum, Y. K.: Pseudohypertrophic Muscular Dystrophy, J. PEDIAT. **3**: 352-358, 1933.
72. Kostakow, S., and Slauch, A.: Deutsches Arch. f. klin. Med. **175**: 25, 1933.
73. Beard, H. H., and Tripoli, C. J.: The Effect of Feeding Amino Acids in Cases of Muscular Dystrophy, New Orleans M. & S. J. **86**: 205, 1933.
74. Harris, M. M., and Brand, E.: Metabolic and Therapeutic Studies in the Myopathies, J. A. M. A. **101**: 1047, 1933.
75. Thomas, K.; Milhorat, A. T.; and Techner, F.: Ztschr. f. physiol. Chem. **205**: 93, 1932.
76. Voshell, A. F.: Progressive Pseudohypertrophic Muscular Dystrophy, South. M. J. **26**: 156, 1933.

American Academy of Pediatrics

Proceedings

FOURTH ANNUAL MEETING OF THE AMERICAN ACADEMY OF PEDIATRICS

CLEVELAND, JUNE 11 AND 12, 1934

Panel Discussion on Dental Caries

Leader: Frederick F. Tisdall, M.D. Assistants: Theodore B. Beust, M.D., D.D.S.; Julian D. Boyd, M.D.; Russell W. Bunting, D.D.S., D.Sc.; Glen E. Cullen, M.D.; Percy R. Howe, D D.S., D.Sc.; Martha Koehne, Ph.D.; and Leo F. Rettger, Ph.D., LL.D.

The Panel Discussion on Dental Caries was called to order at 9 A.M. in the Cleveland Medical Library by Frederick F. Tisdall, M.D., Toronto, Ontario.

CHAIRMAN TISDALL.—There is no disease so prevalent today as dental caries. At the White House Conference it was shown that over 95 per cent of all school children suffer from this ailment. Until recently the pediatrist has not considered tooth decay as falling in his province and has rather looked upon it as being in the field of the dental profession. Recent investigative work by both the dental and medical professions has furnished results which are of great importance to us.

The discussion this morning has been arranged for the purpose of drawing to our attention, so that we can apply in a practical way in our everyday practice, the recent findings which we believe will be of assistance in reducing the incidence of this condition.

It is hardly necessary for me to draw to your attention the fact that if we, as pediatrists, can be of influence in reducing tooth decay, we can also reduce to a great extent the incidence of many chronic and crippling diseases which occur later in adult life.

The Academy is fortunate this morning in having with us for this discussion outstanding members of the dental profession and other individuals who are not members of the Academy. They have very generously given of their time to come here and help us this morning. I am sure we are all grateful to them for this courtesy.

I have asked Dr. Beust, of Louisville, to outline some of the fundamental facts which are necessary for us to know in order to understand the subsequent discussion. With the exception of this address and a few words from Dr. Howe on heredity, the discussion will be entirely informal. Whenever we have a child with tooth decay which occurs in spite of our best dietary means, we always take refuge in saying it must have been hereditary.

DR. THEODORE B. BEUST (LOUISVILLE, KY.).—Two calcified structures of the tooth are commonly involved in caries. Of these, the outer covering, the enamel,

contains but 2½ per cent organic matter. Most investigators regard the enamel as an inert buffer tissue deposited for the protection of the dentin.

The dentin is a calcified intercellular collagenous substance of a composition closely approaching that of bone. In the unmodified state it contains countless open canals called tubules. Each tubule contains a process of one of the surface cells of the pulp. These are known as odontoblasts. Stains and even India ink may be injected from the pulp cavity into the tubules. The property of permeability is disclosed by such an experiment. This in itself indicates the possibility of metabolic flow. Dentin, as a rule, does not long remain in the unmodified state. Under the influence of irritation or age, changes that may lead to obliteration of its tubules may occur. Such modified dentin is regarded as hypercalcified, or sclerosed. Sclerosis may be hyaline or opaque, depending on the nature of the changes within the tubules.

Caries.—Histopathologic phenomena attending an attack by caries are complex, enigmatical, largely contradictory, and withal poorly understood. The classical con-

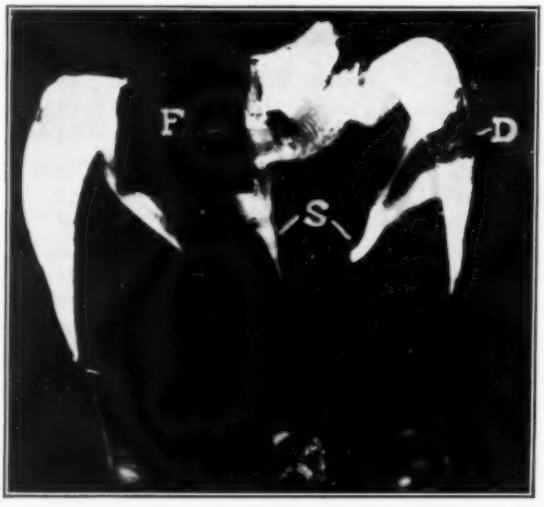


Fig. 1.—Stained ground section. *D*, Incipient decay; *F*, space left by a dislodged filling; *S*, sclerosed, impermeable, chromophobic resistance zones.

cept of the chemicoparasitic theory of caries, as evolved by Leber and Rottenstein in 1860 and enlarged by W. D. Miller in the early '90's, assumes an initial decalcification of the enamel by acids formed by the mixed flora of the mouth. The attack on the enamel, as described by Miller, consists of a dissolution of the constituent elements of its surface. These are washed away whereby a depression, which gradually deepens, forms a cavity. This account describes that rare type of caries characterized by a wasting away of freely exposed enamel surfaces whereby poorly demarcated cavities are formed. It also applies to the phenomena attending experimental caries, which may be induced in test tubes by immersing teeth in fermenting broths inoculated with aciduric bacteria. The localized sharply defined form of caries with which the dentist commonly deals presents another picture. In the latter case the enamel rarely suffers a loss of surface continuity before reactions manifest themselves, often reaching to the immediate neighborhood of the pulp. Enamel, when experimentally exposed to a lactic acid mixture of

a potence comparable to that found in fermenting broth, is easily displaced or dissolves. On the other hand, naturally decayed enamel remains sufficiently resistant to retain its surface continuity during the grinding of the section, as shown in the illustration.

These observations cast suspicions on the belief that lactic acid is an etiologic factor in incipient caries. It is to be understood that this view is not held by all of the members present, particularly Dr. Bunting.

Influence of Environment on Caries.—Oral environment has obviously nothing to do with caries incidence. Caries-free patients with teeth bathed in decomposing secretions and covered with living microorganismal film are familiar to every practitioner. On the other hand our most fastidious patients, skilled in the use of approved accessories to prophylaxis, are commonly unable to combat the ravages of caries. Instances of the decay of only one of two adjoining teeth, which by their position in the arch are exposed to the same conditions, may be found in almost every mouth.

Vitality of the Tooth.—The occurrence of vital phenomena within the dentin can no longer be questioned. This view, however, has long been combated by well-known dental authorities. The contrary views are based on erroneous preconceptions and faulty methods of examinations. Reactions, as can be shown by new methods of disclosure, attend caries, abrasions, erosions, hypoplasias, all kinds of enamel defects, and age changes. The changes induced consist mainly in the sclerosis of the organic parts of the dentin whereby resistant barriers are laid down. In properly stained ground sections, these are visible to the unaided eye.

Enamel Vitality.—Erroneous views concerning structure have long presented obstacles to a correct interpretation and demonstration of changes occurring within the enamel. Howard Mummery, English histologist, showed zones of resistance surrounding incipient enamel caries. These consisted of chromophobic resistance zones which surrounded the point of attack. Such zones actually exist in the large majority of cases.

The indisputable fact that certain spaces between the enamel rods, known as tufts, may be stained through the pulp cavity and dentin of young erupting teeth and that these tufts no longer respond to any known methods of staining after a tooth has matured must be accepted as proof that vital changes occur.

CHAIRMAN TISDALL.—Dr. Beust has brought out the point that decay is not the result of acid products. Dr. Beust has shown evidence that the tooth is a living structure, which fact is important to keep in mind in regard to the remarks Dr. Cullen is to make in connection with the metabolism of the body having an effect on the tooth. The whole presentation of Dr. Beust is simply to introduce the subject to us and familiarize us with some of the terms which will be used.

DR. PERCY R. HOWE (BOSTON, MASS.).—Inheritance in its relation to dental caries is a subject which receives comparatively little attention in dental literature, and there seems to be no reason why it should.

The universality of caries is one indication that inheritance plays but little part in the defense against tooth distintegration.

Another obvious fact supporting the same contention is that races with an inheritance of many generations of sound teeth succumb to caries when they adopt modern foods. So that for the purpose of the present discussion, it might be well to omit the consideration of inheritance.

DR. RUSSELL W. BUNTING (ANN ARBOR, MICH.).—We believe that mouth cleanliness has a relative effect on dental caries. If caries is active in the mouth, the more unclean the mouth is the more decay will result. We have no actual proof of this

view. It is not, however, a determinant because there are so many cases in which we find mouths that are exceedingly dirty, and yet there is no decay. In others, which are relatively clean, we find plenty of decay. So I should say hygiene of the mouth has a relative effect on reducing the amount of caries.

DR. HOWE.—I think with Dr. Bunting that mouth cleanliness is a hygienic measure and as such is commendable. Of its precise relation to caries, I am in doubt, for notwithstanding the fact that oral prophylaxis has been relied on these many years for preventing caries, the incidence has not been appreciably lessened.

We may again refer to primitive races in whom tooth powder and brush are unknown and who are remarkably free from caries. Evidently it is not safe to confine preventive efforts to hygiene alone, but other factors must be considered.

CHAIRMAN TISDALL.—The next point we have for discussion is the question of the bacteriology of the mouth. This brings to mind the statement of Dr. Beust, that aciduric factors on the outside of the tooth have nothing to do with tooth decay.

DR. BUNTING.—I appreciate the effort of Dr. Beust to find some new lead on the problem of dental caries which has baffled all students of that subject for some time.

The initial lesion of caries is the breaking down of the tooth enamel, which is a highly calcified and highly specialized substance. I can think of no way in which this highly calcified substance can be broken down other than by the action of acids.

The peculiar phenomenon in this process by which the lesion progresses into the tooth cannot be produced by any laboratory experimental method. Placing a tooth in acid does not produce the same type of penetration. This phenomenon occurs in the mouth only on vital teeth, and caries in a nonvital tooth in the mouth is precisely like that of the carious lesion produced in the laboratory. Therefore, there are unquestionably osmotic factors involved in caries in living teeth which determine the type of penetration of the acids. We know of no solvent that will break down the enamel substance other than acid.

In the vital tooth the lesion is influenced by the drawing of these acids deep down into the dense layers of the enamel and dentin very rapidly by some force, perhaps an osmotic pull by the tooth pulp as in the interchange of fluids of dissimilar character through a semipermeable membrane.

The action of acids on the enamel is definitely made penetrating by some action in a vital tooth that does not exist in a nonvital tooth. I am unable to visualize any way in which the enamel could be broken down except by the action of acid.

If environment has nothing to do with the action of caries, why should caries almost invariably occur in certain definite locations on the tooth, that are definitely determined by the environment and anatomic relationships of the tooth, and by no other means?

DR. BEUST.—The fact that enamel stands up in incipient caries and does not dissolve away, as when attacked by acid, has been known for years and was also known to Miller. However, no one has attempted to apply this knowledge in our determination of the causes of decay.

Dr. Bunting may be correct in his assumption that the enamel of pulpless teeth dissolves out, as occurs in a test tube in a broth containing aciduric bacteria. I have not observed it. However, the changes in the dentin that occur in caries are to be found only in teeth with living pulps.

The enamel in caries does not dissolve away and has not the appearance of having been dissolved by acid. I am not a biochemist and am presenting phe-

nomena that may be observed in making ground sections of teeth for histologic examination.

CHAIRMAN TISDALL.—Dr. Beust, you simply said Dr. Bunting was not correct in his statement that acid produces tooth decay. You believe it is some other process, but what is that other process?

DR. BEUST.—It could conceivably be a protein, a toxin. It is commonly believed that decay starts under a plaque or colony of bacteria or other microorganisms. For illustration, most cavities start exactly at the contact point between two teeth. These are surfaces exposed to interdental wear. It is not conceivable that a colony of sufficient size to do this damage could flourish there. Inferentially, we have in those spaces conditions favorable to putrefaction and not to acid formation.

I do not mean to imply that acid could not in any case start caries. However, I contend that the appearances, as seen in ground sections, indicate that we are not dealing with erosion from the action of acid.

CHAIRMAN TISDALL.—Dr. Cullen, could you tell us from the chemical standpoint whether acid localized to a point on the enamel could result in dissolution of enamel and how that acid could be kept there?

DR. GLEN E. CULLEN (CINCINNATI, OHIO).—Dentists talk about the dissolving of enamel. Is this histologically a dissolving of the crystals of calcium phosphate carbonate, or is it the solution or disintegration of the very small protein-like material which binds the crystals together? What is attacked in the very first stage of the decay?

DR. BEUST.—If enamel is exposed to a dilute acid, the interprismatic substance (that is the cementing substance between the rods) is first dissolved. If the action is allowed to continue, the acids attack the rod from its periphery. Therefore, at first the rods are loosened and with continued penetration drop out or otherwise disappear. That is what happens when the agent is an acid, and that corresponds to Miller's conception of the action of acid on enamel. In the case of enamel that has been attacked by natural caries, the rods, of which the enamel is composed, the rod striations, and the structures known as lamellae and tufts, remain visible. As already stated, the enamel remains intact and there is no loss of surface continuity.

DR. BUNTING.—We are dealing with two substances in the enamel, one a very highly calcified rod, the other a substance between the rods of a different nature, which we speak of as cement substance. Enamel is from 95 to 97 per cent inorganic material and not more than 2 to 3 per cent of organic material on which any proteolytic action may occur so that the breaking down of the enamel must be almost solely a process of breaking down of inorganic material.

If you apply acid to the ground surface of a section of enamel or to the exterior of the enamel, the interprismatic substances are first attacked, and gradually the rod is dissolved somewhat later.

It is an interesting observation that when the acid of caries attacks the enamel of a vital tooth it follows along the interprismatic substances and may travel deep down to the dentinoenamel junction and seriously affect the dentin while the enamel rods are still standing. If you place a tooth in acid, the rod and the interprismatic substance are dissolved simultaneously, producing an open cavity as Dr. Beust has said. But the same thing takes place in the dead tooth. I have seen many times in sections of caries in the dead tooth a mass decalcification similar to experimental caries. There was no initial deep penetration of the enamel. Therefore, it seems likely there is a vital action which carries the acid in toward the pulp in vital teeth.

I should like to ask whether there is any conceivable way by which such a highly inorganic material can be broken down other than by acid decalcification.

DR. CULLEN.—You point out that the amount of organic material is very small. That does not mean, of course, that the cement is probably of the nature of mucoproteins. It seems to me the very fact that the cementing material goes out first would indicate that you should look for some substance which dissolves it selectively. Does the acid of which you are thinking dissolve that material?

DR. BUNTING.—The initial acid penetration of the enamel follows the interprismatic substance. The organic substance in the enamel consists of a sheath around the outside of the rod. Between these rod sheaths there is inorganic material, a cementlike inorganic substance which can be dissolved with acid more readily than the enamel rod. If you take a ground section of enamel, apply acid to it, and wash it off, you will find the first effect has been on the interprismatic substance and less on the prisms. It will cause the prisms to stand out in bold relief, a common method of preparing histologic sections of teeth. Acids will disintegrate the enamel in a manner quite comparable to caries in the nonvital tooth.

DR. CULLEN.—While I do not question that acid could do it, why is the acid the only thing that could do it? I think in this discussion we ought to be quite clear what we mean by acid. Do we mean hydrogen ion concentration of the saliva which fixes the local environment of the teeth, or do we mean the concentration of free lactic acid? My impression is that the saliva is a highly buffered material. How can there be enough free acid present, in such a buffered solution as saliva, to cause acid solution of the enamel?

DR. BUNTING.—We can only explain dental caries from the standpoint that the acids of caries are formed locally on the tooth and are protected against the buffer action of the saliva. Otherwise there would be no caries; if the saliva could reach every surface of every tooth continually, there would be no caries.

CHAIRMAN TISDALL.—Dr. Bunting, from the biochemical standpoint, do you believe it is possible for a mucoplaque to wall in bacteria with the production of acid there and that acid attacks the tooth without attacking the mucoplaque and dissolving it?

DR. CULLEN.—In attempting to get at the mechanism by which enamel is disintegrated, I feel we should not limit ourselves to thinking only of the effect of acids. We should consider the possibility of the solubility of enamel by many other agents than acids.

Dr. Bunting said that he could not conceive of any process whereby this enamel could lose its structure except by acid solutions. In the living organism, bones lose their structure. They become decalcified following various experimental procedures in which there can be no question that the bones are not in an acid environment.

Years ago Dr. Gies observed that the mucin which was precipitated on the teeth was much more soluble in weak acids than in weak alkalis. If he used weak alkali, he had a slimy mass from which we get the word "mucin." When he used acetic acid or citric acid, he got a solution of mucin. If there is a tiny walled-off area in which there is a colony of bacteria producing acids away from the neutralizing effect of saliva, what is the possibility of acid dissolving the mucin at the same time? Further, if you have a walled-off space like that and acid dissolves calcium carbonate phosphate crystals of the enamel, why does not that calcium dissolved out from the tooth immediately neutralize the acid?

DR. BUNTING.—You have asked some very pertinent questions which cannot be answered at the present time.

The question you raise refers to an operation which occurs in a very minute area on the tooth and involves some very minute biochemistry, which so far as I know, no one has been able to study or evaluate. We have tried to find out what could break down the tooth. Acid, that is, a solution of high hydrogen ion concentration, has the ability to combine with the enamel and to put it into solution either by degrees or in mass, depending on the rapidity of the acid production or on the vitality of the tooth.

Where does the acid come from? Certainly not from the saliva. We can only conceive of this acid as having been formed in definite localities on the tooth, otherwise the entire tooth would be affected. There is every reason to believe that the anatomy and environmental relationships of the teeth very definitely determine the location of caries on the tooth. At the present time we can only see caries as being a destruction of the tooth by a very localized process, in which acids are formed presumably by bacterial action on carbohydrates.

DR. CULLEN.—There is where I take issue. In the body, certainly, we have breaking down of the greatest variety of both inorganic and organic compounds. We are talking about the organic cementum in this tooth.

DR. BUNTING.—Is there any evidence that the initial step in caries is the destruction of the organic matter in the enamel? I have no such information.

DR. HOWE.—We need to know more about calcification before a definite theory of caries can be evolved. In the laboratory I have introduced aciduric bacteria into the diets of animals, and no caries followed in any instance. On the other hand, caries has developed among my deficiently fed animals, especially monkeys.

A well-known phenomenon which often occurs seems to militate against the fermentive theory of caries. This consists of the hardening of the floor of the carious cavity to the extent that the bottom becomes hard, polished and flintlike. Miller calls this healed decay, and it occurs in the face of bacterial growth. It is this process which has been induced by controlled feeding, which at the same time appears to bring about a general reduction in the appearance of new caries.

Dr. Boyd has reported cases of this description, while at the Forsyth Dental Infirmary we have had similar results in a group of about 150 cases in which an estimated reduction of nearly 80 per cent occurred. Compared with results in a group of about the same size, the reduction in caries by dietary regulation was distinctly greater than that effected by usual hygienic and reparative procedures. Many other similar reports are on record, and their number is increasing.

While it is said by some that tooth structure does not enter into the question of caries, this seems a purely empirical conclusion since by microscopic examination it is difficult to find structurally perfect teeth among the children of today.

It is evident to anyone who sees daily the condition of the teeth of children in large clinics that the assistance of the pediatrist is greatly needed in the matter of the growth and development of sound, rugged teeth and of the bones which support them.

The temporary teeth begin to calcify somewhere about the seventeenth fetal week, and the crowns are practically complete at birth, while the permanent teeth begin to calcify shortly before birth and are only fully complete at the time when general bone growth is complete, or about the age of twenty years. Thus the character of the teeth is in the hands of the pediatrist three or more years before the dentist sees them. Through his scientific knowledge of food, the pediatrist can do much toward the elimination of caries and toward the stimulation of normal bone development.

Experimental studies show that the development of sound, regular teeth goes hand in hand with normal growth and development as a whole and, conversely, that

tooth derangement is symptomatic of a trend of irregular metabolic conditions, seemingly insignificant at first, or perhaps unrecognized, which, when continued over a sufficiently long period, cause pathologic conditions similar to many of those found in middle life. It would seem both from clinical observation and laboratory investigation that dental caries may well be considered as one of the early symptoms of nutritional disturbances in which mineral and vitamin deficiencies play a considerable part.

CHAIRMAN TISDALL.—It looks as though we are still uncertain as to exactly what the process is which goes on in the production of tooth decay. We should summarize this phase of the discussion to get on to the parts which may be of more interest to pediatricians.

DR. BEUST.—It is generally conceded that the entrance of the caries agent occurs in the tissue between the rods. The cementing substance that holds the rods together is of higher organic content than the rod itself. Gottlieb, of Vienna, stated that caries commonly enters the tooth through a lamella. The lamellae are structures which lie between the enamel rods which in youth at least (when caries is most rampant) are made up largely of organic matter. We have here also an indication that the agent bringing the enamel to dissolution need not necessarily be an acid but could conceivably be a proteolytic toxin.

DR. CULLEN.—A disintegration of enamel occurs at the surface of the tooth in dental caries. This has to be considered as a solution either of the enamel as a whole or of some part of the enamel structure. The breakdown of organized structures that occurs in living organisms is brought about through the agency of enzymatic processes. The building up and breaking down of all types of tissue, muscle, tendon, and bone are not functions of acid or alkali in themselves, but of specific enzymatic processes. Acid and alkali may influence the nature of these processes but only indirectly by their effect on the enzymatic action. When we study your work, Dr. Bunting, and see your evidence of the relation of definite types of bacterial flora to tooth destruction, we are forced to think that if certain types of bacteria can either initiate or hasten enamel disintegration, the chances of their doing this by means of the enzymes they liberate are tremendously greater than through the one substance—lactic acid. Even then the term "acid" is most misleading, for either in saliva or in the presence of dissolving calcium salts the lactic acid produced must be present as the lactate salt.

CHAIRMAN TISDALL.—I think we had better go on to another phase of this discussion and simply summarize the situation by saying that teeth do decay. How, we do not know. We have some of the most eminent members of the dental profession here and they cannot agree among themselves. It appears that acid may be a factor, or a putrefactive process may be a factor, or something else. Fortunately they have an open mind, so let us maintain an open mind in our thinking for the next hour.

Dr. Bunting has done a great deal of work in support of the contention that it is acid and that acid is produced by a certain type of bacteria. He has conducted experiments over many years on children. Could you present the essential conclusions you have come to from your work? Then perhaps Dr. Rettger will discuss it from the bacteriologic standpoint.

DR. BUNTING.—I am not a bacteriologist, a chemist, nor a nutritionist, but I am associated with individuals who are well qualified in these respective fields and who are cooperating with me in the study of this problem. In the beginning, we looked for some chemical, bacteriologic, or immunologic difference between individuals with decay and individuals without decay; we have spent five years in

searching for that difference in carefully selected individuals. The only constant differential we have found is the presence and the activity of growth of an organism which we and others call *B. acidophilus* because of its similarity to the organism of that name living in the intestinal tract.

We have seen that organism growing luxuriantly in the mouth of every individual who had caries, and in those who had no caries the organism is either not present at all and cannot be made to grow in the mouth even when it is implanted there daily, or it is present only in small numbers.

This is the only differential thus far demonstrated. I believe there are some other differences which as yet we have not found that determine whether or not organisms can grow in the mouth. I believe ultimately we will find some chemical difference in the saliva or the tissues of the individual who has caries from that of the one who has not.

Dr. Howe has said there is nothing in heredity and that all people have dental caries. This statement is not quite true. Five per cent of all people do not have dental caries. You, yourselves, know individuals in your own practices who have lived all their lives and never had decay in their teeth. They are different from the 95 per cent of the people who have caries. What is that difference? Did they inherit it? I do not know, but it is an individual characteristic which marks them as different in regard to this disease from the 95 per cent. Over on the other end of the scale there are from 5 to 10 per cent of individuals who are extremely susceptible to caries. Do what you will or feed them what you like, they will have caries. I do not believe that Dr. Boyd claims that he gets 100 per cent control of dental caries. If he does, I shall be disappointed.

DR. LEO F. RETTGER (NEW HAVEN, CONN.).—During the last two years of study of mouth and teeth bacteria, our laboratory has had the cooperation of the dental division of the Department of Pathology at Yale University; we hope that, when we publish our results, they will not be regarded by the dental profession as entirely those of mere laymen.

We passed altogether over the question of strength of acid (or hydrogen ion concentration), which is a very important aspect of the subject. Can a small amount of acid or a condition of weak acidity initiate decay, or is relatively strong acid required? We must assume that conditions in the mouth are such that there cannot be any very appreciable acidity in any part of it, certainly for any length of time. There are perhaps localities around the teeth and in defective teeth which are more or less acid at times. Whether enzymes or other agencies take an active part in the decay, I will not hazard to state.

In the mouth, where there is abundant food material for bacteria and favorable hydrogen ion concentration, bacteria must be looked upon as the sole producers of acid, if and when an acid condition is brought about. The saliva is highly buffered and neutralizes all or much of the acid that is formed. Some have assumed that free acid is of common occurrence in the mouth and have asserted that *L. acidophilus* is responsible for the acid. I protest against such an assumption or claim, which is unwarranted by any existing evidence. Why not refer to acid-producing and acid-tolerating bacillary forms as aciduric bacilli or lactobacilli to distinguish them from mouth streptococci which also are aciduric or at least moderately aciduric? The lactobacilli are rod-shaped organisms; they may be short or long and occur singly or in long or short chains. They are gram-positive, produce, and tolerate appreciable amounts of acid, hence the term "aciduric."

The lactobacilli constitute a complex genus, different members of this genus, often varying widely from each other in very important respects. Why not refer to the mouth lactobacilli as such, as *Lactobacillus buccalis*, or by some other name, rather than *Lactobacillus acidophilus*? The lactobacilli of the intestine constitute

a definite, different type. We have been engaged in studies of the intestinal lactobacilli in my laboratory for some twenty years and have spent at last five years on the lactobacilli of the mouth. There is as big a difference between the types present in the mouth of those occurring normally in the intestine as there is between the typhoid bacillus and the ordinary colon bacillus, or between the anthrax bacillus and ordinary *B. mycoides* of the soil and water. A certain investigator once claimed that he had found anthrax bacilli in abundance in the dust and seed of an ordinary hayloft. He may have been correct in his claim, but the chances are that the so-called *Bacillus anthracis* was nothing more than *Bacillus mycoides*, which is not a disease-producing organism. I mention these examples to impress upon you that organisms may appear alike in many ways, yet differ from each other in certain important respects. The same reasoning may be applied to the lactobacilli of the mouth and of the intestines.

In stressing the question of numbers of lactobacilli in the mouth I am entering into a conflict with others, particularly Dr. Bunting. Some four years ago a medium was described by Rodriguez, namely ordinary infusion agar containing 10 per cent horse serum, on which he plated the viable organisms of the saliva. He stated he could easily demonstrate one million or more *L. acidophilus* per cubic centimeter of saliva, especially of carious mouths. We made up the medium and used it in the manner described by him. We found often, as he did, as many as one million gram-positive bacteria per cubic centimeter of saliva, but instead of their being lactobacilli, as he claimed, they turned out to be streptococci.

There is frequently so much resemblance between the lactobacilli and the streptococci of the mouth morphologically and culturally that, unless one is keenly on the alert and well trained in such observation work, the real differences will go unrecognized.

We are of the belief in our laboratory that the lactobacilli of the mouth may at times be relatively numerous, but their incidence in carious and in noncarious mouths is relatively low compared with that of certain streptococci, which we have no difficulty in finding in very large numbers in the saliva and on the surface of teeth, both normal and decayed. By scraping the coating on the teeth with glass scalpels, weighing this material, and determining bacteriologically the number of bacteria removed with this scraped material, we found the streptococci almost invariably to be the predominating organisms and the lactobacilli in insignificant numbers, as a rule, and at times apparently absent. In the cavities themselves we quite frequently observed lactobacilli, but they were found to be most numerous in the older portion of the cavity, while the streptococci were particularly abundant in the layers of the decay material.

We have approached the question of comparative incidence of streptococci and lactobacilli from various angles. As already stated, we have made a study of the saliva itself and of scrapings of the teeth and decay material from the cavities, to determine the numerical importance of lactobacilli and their possible relationship to caries. We find lactobacilli in the mouth and in tooth cavities, as a rule in relatively small numbers, but they are definitely oral types, and differ materially from those of intestinal origin. We are as yet unable to establish any definite correlation between their occurrence and dental caries. Why not give the streptococci some consideration, because they are practically always present in such large numbers in the saliva, in the coating on the teeth, and in other parts in and around the teeth?

The mouth streptococci do not produce as much acid in glucose broth and other carbohydrate media as do the lactobacilli of the mouth. However, the maximum acid production by the mouth lactobacilli is only slightly greater than that of the prevailing mouth streptococci, which has been designated *Streptococcus mutans* by Clark and regarded by him as an important factor in dental caries. A difference

in acid production for a few tenths in terms of hydrogen ion concentration of these two aciduric organisms cannot have any real significance, particularly when we consider that the hydrogen ion concentration of the mouth or any part of the mouth, including the teeth, cannot be high at any time.

In taking issue with some of my contemporaries, I do not wish to underestimate their work, but I am confident there are many things yet to be learned regarding the etiology of dental caries. Indeed, you cannot but conclude from the discussions here that there is little as yet concerning dental caries about which investigators are agreed, and that there is a long road still ahead of us. Let me mention here one phase of bacteriologic study which has been almost completely neglected, namely that of the anaerobic bacteria and their possible rôle. What part, for example do the leptothrix-like forms, which we have at times observed in abundance in and on teeth, play, not saying anything of numerous other species and types of anaerobic bacteria which can and do find the mouth a favorable habitat?

DR. BUNTING.—Not being a qualified bacteriologist, I can only say that the organisms which we call *B. acidophilus* are very definitely related in their growth to dental caries. From our statistics covering hundreds and hundreds of cases, this relationship has very definitely appeared.

The distinctive aciduric characteristics of *B. acidophilus* and the correlation of the quantitative studies which we have made have revealed an almost spectacular association; therefore, we have been led to view it with interest. Whether this bacterial factor is the all-controlling influence and is the answer to the question, we are not certain.

DR. JULIAN D. BOYD (Iowa City, Ia.).—We are fortunate in having available for study a group of over a hundred children who receive weighed prescribed diets, and who adhere to these quite rigorously as a group. This type of diet regulation has proved adequate to control and arrest caries. Individual children who have had reactivation of caries or delayed arrest have been those who flagrantly failed to follow the prescribed regimen. After a survey of the factors which might be credited with the protective effect upon the teeth, various bacteriologic, biochemical, and metabolic studies have been made during the past eight years. In reference to the bacteriologic phase now under discussion, the report of our findings, which appeared recently, offered evidence that acid-producing bacilli and other organisms of high acid-producing ability exist in large numbers in the mouths of children receiving protective diets, whose mouths have been shown by repeated dental observations to be free from active caries. Moreover, children with active caries, placed on such a dietary regimen and observed during the period of inactivation of caries, show no characteristic change in the morphologic nature of the oral flora or in their ability to produce acid in significant amounts. The incidence of such acid-producing organisms appeared to be as great in frequency and in numbers in the mouths of children receiving continuously such protective diets as in the mouths of those whose diet was not supervised. The validity of these conclusions has been questioned by other workers in this field on the basis of the technic employed, which was not considered of quantitative value in estimation of aciduric rods in the oral flora. Because of this, further observations have been made, in which the method described by Hadley was employed in estimating the number of aciduric rods present, and electrometric methods were employed in estimating the degree of acidity attained in the culture media after intervals of 50 and of 100 hours' incubation. The results obtained have not led to change of the conclusions reported in the previous study. We found a higher average incidence of so-called *B. acidophilus* in the mouths of those free from active caries and a lower number in those with active caries than had been reported by Dr. Bunting's coworkers. Our

data reaffirm the value of diet regulation in the control of caries and offer nothing to indicate that the mechanism is one of inhibition of growth of aciduric organisms.

DR. BUNTING.—In the bacteriologic survey of those groups which we have had under dietary control and in which we have practically controlled caries, we have found that those children who had a cessation of caries also had an almost complete disappearance of *B. acidophilus*. *B. acidophilus* either disappeared or became very low in count in practically all cases of arrested caries.

This year we are going to make a survey each week of a large group of orphanage children to obtain further data. At the present time we have very definite proof that diet does change the bacteriology of the mouth, and we believe it possible that this is the reason why caries is arrested.

CHAIRMAN TISDALL.—The next part of the discussion will probably be of the greatest interest to us as pediatrists, namely the influence of diet on tooth decay. I shall ask Dr. Boyd to start this part of the discussion. We must remember there are many different factors. It will be best if we discuss a few of the individual factors first and the diet as a whole later.

DR. BOYD.—While our first reports of arrest of caries in children were based upon observations of diabetic children, subsequent studies established that similar results are obtainable with nondiabetic children and that the control appears dependent upon the degree of completeness of the diet in the various essentials for normal nutrition. Results of similar nature have been obtained with diets of widely differing content of carbohydrate and of fat. Arrest of caries is not accomplished with equal ease in all children; with some, a moderate regulation of the diet is all that is necessary, while with others the most scrupulous dietary supervision is required to arrest caries. While Dr. Bunting interprets this varied degree of susceptibility on the basis of the nature of the mouth flora, our data do not indicate the importance of this factor but indicate rather that it is dependent upon the relative efficiency of the child's metabolism under given conditions.

We have held throughout that the integrity of the tooth paralleled that of the organism as a whole and that undue weight should not be placed on any one factor of the diet in the control of caries. Efforts should be made to offer the most suitable diet in all of its phases and consideration directed to the child as a whole rather than to his teeth. Studies of the average American dietary indicate that too few of several of the essentials for normal nutrition are included. While the deficit of some of these is greater and more constant than that of others, none should be ignored in this connection.

DR. MARTHA KOEHNE (ANN ARBOR, MICH.).—We have studied two groups of children, one living in an orphanage, in whom the incidence of decay over a period of years has been remarkably low, the other on children living at the University Hospital at Ann Arbor. These children were confined to the hospital because of acute gonorrheal vaginitis. Aside from the first few weeks of their stay when they had a marked polyuria, their metabolic processes were normal. The children did not appear to be sick.

We made up the menus, which were very acceptable to the children and were nutritionally satisfactory. Every portion of food eaten by the children was weighed throughout their long stay in the hospital. We allowed free choice as to the amounts of food eaten from the menus available. Each child had the opportunity to eat as much as he wanted of available food.

It is remarkable how constant the food intake was over periods of weeks and months. Some of the children stayed as long as a year and one-half. They ate very well. The only modifications we made of the basal diet were, in some instances,

to allow the children to have as much candy as they could eat as extra food. The children cooperated with us very well in continuing to eat the basal diet satisfactorily after the candy was added to their diet, even though some of them ate as much as a quarter to a third of a pound of candy every day for a year. They stayed in good general health throughout the periods of observation.

Chemical studies were made on the blood serum and saliva of each child once a month. Balance studies were made at different intervals during the stay of a selected group of the children to determine if retentions of nitrogen, calcium, and phosphorus were different during periods of high candy feeding in comparison with periods of low intake of added sugar. Results showed practically no difference in the retention of calcium and phosphorus resulting from high sugar intake. There was a slight reduction in the retention of calcium, but it was too slight to be of significance. Phosphorus retention was slightly increased and nitrogen retention was definitely increased.

During the time the children were under observation, dental examinations were made every three months, including bite wing x-rays. All results were then correlated for the respective children during the long period of time they were under observation.

Mrs. Mellanby's claims notwithstanding, the orphanage children, among whom the incidence of caries was remarkably low, ate almost half of their calories from starch-rich food. They had no butter for a period of five or six years, milk for breakfast only and very little meat. The very desirable dental condition was maintained on a diet which would be regarded as undesirable for growing children.

In the hospital group the children were on an optimum diet for the time they were under observation. Differences consisted in the proportion of calories from artificially sweetened food. The children can be divided into several groups for purposes of discussion.

We had fourteen children on the basal diet alone for the length of time they were in the institution. Nine of these developed no dental change during that period although all had open cavities on admission. Among these nine children, the particular aciduric organism we have been discussing either disappeared completely from the mouth or was there in almost negligible concentration during the last few months of observation.

Five of these fourteen children, in spite of following diets that were very satisfactory nutritionally and that were low in added sugar, continued to develop cavities and the concentration of aciduric organisms remained high.

There were thirteen children who, for varying periods of time, were given extra candy *ad libitum*. Three of these children had never had cavities in their teeth, in spite of having been on home diets which were deplorable nutritionally. They continued to eat our basal diet very well after being given candy, and studies showed they retained practically the same amounts of calcium and phosphorus as when on the basal diet alone. These three caries-free children remained negative to the aciduric organisms and developed no cavities during the periods of high sugar intake. Seven other children, however, continued to have active caries, and the longer they were on the high sugar diet, the greater the amount of dental change observed at each successive examination. Aciduric organisms of the lactobacillus group increased rapidly in their mouths.

There were several of the orphanage and hospital children who seemed to be exceptions. With the orphanage children on the inadequate diet, the great majority of those studied were negative to the aciduric organism in the plates studied by the Hadley method. There were a few, from 20 to 30 per cent, who continued to have cavities develop and to show high counts of this aciduric organism.

We found no differences in the calcium and phosphorus content of the saliva that could be correlated with caries immunity, caries inactivity, or caries susceptibility. For the hospital children, whatever the calcium and phosphorus content of the saliva was on admission, it remained rather constant on our satisfactory diet. The acid neutralizing power of the saliva, as measured by the CO_2 capacity, remained constant for the individual regardless of marked changes in basic value of the diet and in caries suspectibility.

We did find that reducing the proportion of caloric intake from added sugar brought about a marked reduction in caries activity in the majority of the children having an average susceptibility to tooth decay. Associated with this lowered caries susceptibility was a marked reduction in the concentration of the aciduric organism. The reverse was also found to be true except for those who were very susceptible to tooth decay.

DR. BOYD.—In a recent correlation of various chemical and metabolic studies of children who had been seen repeatedly in our dental clinic, data on about 100 children were analyzed. We found, as has been reported by several other workers, that no correlation exists between the levels of calcium and of phosphorus in the saliva or in the blood serum and the degree of caries activity in the teeth. Even in a group of children with persistently lowered levels of serum phosphorus, there was no parallel increase in the susceptibility of the teeth to decay. When we studied the retentions of calcium and phosphorus under standardized dietary conditions, however, we found a distinct correlation between the ability of the child to retain these substances and their freedom from caries. Dividing a group of ninety-eight children into those with no cavities in their teeth, those with inactive caries, and those with progressive caries, the average daily retention of calcium per kilogram of body weight was 17 mg. for the first group, 15 mg. for the second, and 10 mg. for the third. The corresponding average phosphorus retentions were 14 mg., 11 mg., and 9 mg. We wish to emphasize that the retentions of those with active caries was fairly in accord with the usually accepted norms; the markedly higher retentions of those free from caries suggests a causative relationship between liberal retentions and tooth resistance. All children in this study were on similar diets, designed to be optimum so far as present knowledge can determine.

Our studies point at present toward a positive correlation between the metabolic integrity of the individual as a whole and his resistance to caries. This seems in agreement with Dr. Beust's observations that sclerosis of the dentin offers resistance to decay. The well-nourished child should have teeth which show the sclerosis of the mature tooth at an earlier age and should be able to throw out protective sclerosis more readily than the child whose teeth are more poorly mineralized.

DR. RETTGER.—The lactobacilli and the prevailing streptococcus of the mouth, Clark's *Streptococcus mutans*, are at times very much alike in their colony form and have similar staining properties, both being gram-positive in young culture. Both are aciduric. It is not always easy to distinguish them, especially since these streptococci at times appear like rod forms. As stated before, we used Rodriguez' 1930 medium and method for making a quantitative study of aciduric mouth organisms generally. We also tried to repeat Mrs. Hadley's observations on the medium she reported recently, and here again we failed, as a rule, to find many lactobacilli mouths both with caries and without caries; but on continued incubation of the plates, we observed numerous streptococci which were apparently similar to Clark's *Streptococcus mutans*, which he described in his 1924 publication and regarded as an important etiologic factor in caries. He emphasized the varying cell morphology of this organism.

I am not satisfied that the correlations between lactobacillus occurrence and caries which have been claimed by some investigators have been definitely established. I am not opposed to the frequently advanced theory that bacteria constitute a factor in dental decay. Indeed, I would not be surprised if they do play a very material rôle, either primary or secondary. I believe, however, that definite evidence is still lacking as to the real significance of any one species or type as a specific agent in the production of caries.

DR. CULLEN.—There are several things which have not been discussed fully. The first is that there is no evidence of any kind that the acid-base balance of the blood is involved in this situation. There are a great number of papers in which attempts have been made to correlate CO_2 capacity, pH of blood, and calcium and prosphorus value of blood with prevention or arrest of dental caries. I believe that the acid-base balance of the blood itself is not the only factor involved. There may be deficiencies of other kinds. First, there is the question of the influence of the vitamins. It is unfortunate that our Chairman has not told about his own work. He has had some important evidence of the value of properly supplementing the vitamin contents of food.

Second, there is the argument in regard to the mineral ash of food. We hear that one should feed a basic ash or an acid ash. There is definite evidence that an excess of acid ash in the food has set up conditions which help the initiation of dental caries. I believe the effect of an acid ash is perhaps a deficiency of base material rather than excess of acid itself.

DR. BUNTING.—It must be quite apparent that the problem of dental caries is exceedingly complex and is still unsolved. Through a grant from the Children's Fund of Michigan a group study of dental caries has been carried on in Michigan during the past four years by highly trained investigators. They have given considerable attention to the establishment of certain basic facts as a foundation for further studies. Their findings have been reported in detail without any definite conclusions being drawn.

Some of the findings of this group are the following: No evidence could be obtained that the calcium-phosphorus intake or the amount of these substances in the blood or saliva has any direct relation to dental caries. No characteristic difference between the bloods or salivas of caries-free and caries-immune individuals has yet been found other than the presence and degree of activity of that organism in the mouth which is commonly designated *B. acidophilus*. As to the dietary control of dental caries, we have observed remarkable instances of arrest of dental disease resulting from the adoption of well-balanced low-sugar diets, but we are not clear as to the manner in which this change is produced. We know of no particular food factor which has been proved to possess any specific protective action. It is interesting to note, however, that in practically all feeding experiments, irrespective of the particular motif employed, a uniform, fairly adequate and low-suger ration was fed. The majority of children thus fed showed a definite decrease or arrest of dental caries. The only factual conclusions we have been able to deduce from all the dietary experimentation thus far reported have been that a large percentage of children can apparently be protected from dental caries by the adoption of a variety of diets having the above general characteristics but that for many particularly susceptible children no diet yet devised will completely arrest dental disease.

DR. KOEHNE.—I am not at all convinced that a complete adequacy of the diet is an absolute necessity in the control of caries. I am not arguing that children should be given inadequate diets, but if these orphanage children on an un-

satisfactory diet are singularly free from tooth decay, it should be possible to provide adequate diets that are equally successful, once the controlling factor or factors are determined.

Among the group studied by Dr. Boyd, the ages ranged from two to twenty years. There is an enormous difference in the calcium retention of children in these wide age ranges. The children on whom we ran balance studies were from seven to thirteen years of age. In their study there were some negative balances in the caries-inactive group and some very high positive retentions in the caries-active group. If the degree of positive retention is the controlling factor in caries susceptibility, how does he explain such wide individual differences?

CHAIRMAN TISDALL.—In summarizing this very interesting discussion of two hours, I would just like to draw to your attention the following points. Dr. Beust has given evidence that the tooth is a living substance; therefore, we have evidence that it may be affected by the metabolism of the body.

The second point brought out is that hygiene of the mouth has an effect on tooth decay but is of secondary importance.

The third point is that heredity may or may not be a factor but still we can influence the incidence of tooth decay in most cases.

In regard to the fourth point, the process of decay, it is clear that this question is not entirely settled. However, this discussion has served its purpose of bringing to our attention the very complex problems which we have before us and the necessity for further research and further work not only on the part of the dental profession but also on the part of ourselves.

The next point is that the bacteria of the mouth, whether Dr. Bunting's *Bacillus acidophilus* or Professor Rettger's numerous organisms, may or may not have an effect on tooth decay.

The last point is the question of diet, and Dr. Koehne has brought out the possible significance of an excess of sugar in the production of tooth decay. She has also brought out the fact that on a certain poor diet which resulted in diminished body growth, tooth decay did not occur to any degree. However, we as pediatricians are not going to apply that poor diet in our practice.

On the other hand, Dr. Boyd has come along with very positive evidence that a diet which is rich in minerals and rich in vitamins, and has a reasonable distribution of protein, carbohydrate, fat (this being of secondary importance), minerals, vitamins, will reduce tooth decay. Dr. Howe's recent work published in the *American Journal of the Diseases of Children* showed that when the diet is rich in milk, meat, eggs, vegetables, and fruit, which diet supplies particularly calcium and phosphorus and through the eggs some vitamin D, there has resulted a very definite reduction in the incidence of tooth decay.

I think we can conclude by stating that if we pay more attention to this question of tooth decay and use the well-balanced diets, rich in minerals, and rich in vitamins (if I may take the liberty of stating from our own work, possibly vitamin D) that we will go a long way toward reducing the prevalence of this almost universal disorder. If this discussion has served to stimulate our interest for further study in this problem, I think it has served its purpose.

I am sure the Academy is more than grateful to these gentlemen, who are not members of the Academy, for coming such long distances to help us with this discussion.

FOURTH ANNUAL MEETING OF THE AMERICAN ACADEMY OF PEDIATRICS

JUNE 11 AND 12, 1934

Round Table Conference on the Acute Abdomen of Childhood

Leader: Dr. Herbert E. Coe, Seattle, Wash. Assistants: Dr. Philip F. Barbour, Louisville, Ky.; Dr. Clifford Sweet, Oakland, Calif.; and Dr. Philip H. Sylvester, Boston, Mass.

The Conference on The Acute Abdomen of Childhood was called to order at the Wade Park Manor Hotel, Cleveland, Ohio, Monday morning, June 11, by Dr. Herbert E. Coe, chairman.

CHAIRMAN COE.—I consider the expression, "acute abdomen of childhood," medical slang and, like any other slang, it means one thing to one person and another to another. So at the outset it will be well to define "acute abdomen" as meaning abdominal symptoms such as pain, tenderness, nausea and vomiting, irregularity in the function of the bowels, with variations in these symptoms developing suddenly in any previously well child; congenital conditions, such as atresia, are excluded.

In this discussion, may I suggest that we follow two underlying themes. The first and principal theme is that no matter how definitely this term originally had surgical implications, the acute abdomen of infancy and childhood is not necessarily a surgical condition. We may have just as acute and often more acute abdomens in childhood from medical conditions as from surgical conditions.

Many acute symptoms may be relieved by improving the physiologic functions; we call these medical conditions. Other abnormal conditions due to structural or organic changes may require mechanical interference or treatment to restore them to normal; we call those surgical conditions. If we consider the acute abdomen purely surgical we see only one side of the picture and in many instances the terminal side.

The second theme is the fundamental physiologic basis of symptoms in the analysis of disease entities. Symptoms are the normal and constant physiologic responses of the organism to abnormal stimuli, and the point of application and the intensity in the character of the stimuli or the specialized tissues which are involved vary the details of this basic response to produce what we term the diagnostic picture of the disease. Differential diagnosis is a test of our ability to interpret properly the picture presented by fundamental physiologic reactions as they are influenced by local modifying factors. While these are of course self-evident facts, it does us no harm to have the basic physiology recalled.

May I outline the general physiologic concepts underlying the conditions which we are to discuss.

The outline will recall to your minds the usual signs and symptoms associated with acute abdominal conditions.

COMMON SYMPTOMS AND SIGNS OF ACUTE ABDOMINAL CONDITIONS

Anorexia	Local and general pain
Nausea	tenderness rigidity
Vomiting	Pallor
Variations in peristalsis	Sweating
Changes in secretion and absorption	Restlessness
Disturbed acid-base balance	Weak pulse
Rhythmic or paroxysmal pain	Low blood pressure
	Fever
	Leucocytosis

The next outline shows these signs and symptoms grouped as to their physiologic basis.

SYMPTOMS	PHYSIOLOGIC BASIS
Anorexia Nausea Vomiting Variations in peristalsis Changes in secretion and absorption Disturbed acid-base balance Rhythmic or paroxysmal pain	Reaction of the neuromuscular and secretory mechanism of the bowel to visceral nerve irritation
Pallor Sweating Restlessness Weak pulse Low blood pressure	Reaction of the general vascular system to marked visceral nerve irritation
Local and general pain	Reaction of the sensorium to visceral nerve irritation
Local and general tenderness	Combined sympathetic and somatic reaction
Local and general rigidity	Somatic reflex
Fever	Systemic reaction to infection
Leucocytosis	

Abnormal conditions stimulating the physiologic responses called symptoms may occur in various places within the abdomen, and the response will vary accordingly. They may occur (1) within the intestine, and the response will be an increase in the normal activities of the intestine, both secretory and muscular; (2) in the intestinal wall, and the response will be an inhibition of the sympathetic nervous mechanism; (3) on the peritoneal surfaces, and the response will be a stimulation of the sympathetic nervous mechanism; or (4) within a solid viscus, and there will be no response, except perhaps a secretory disturbance, until the process reaches the capsule or peritoneal covering.

I have used the terms "stimulation" and "inhibition" of the sympathetic mechanism somewhat arbitrarily, subject to criticism by the physiologists. It is

contended that a negative or inhibitory reaction is produced by stimulation of the parasympathetic set of nerves. It has helped me, however, in interpreting intra-abdominal reactions, to bear in mind a stimulatory and an inhibitory syndrome.

The stimulatory syndrome:

Sympathetic stimulation:

1. Stimulates the brake mechanism, causing distention
2. Raises the sensitivity threshold, slowing reflex responses, and reducing perception of pain.
3. Increases local vasoconstriction, slowing the visceral circulation, and increasing congestion
4. Increases the tonicity of the sphincters.

The inhibitory syndrome:

Sympathetic inhibition:

1. Inhibits the brake mechanism, increasing peristalsis
2. Lowers the sensitivity threshold, increasing reflex responses
3. Inhibits local vasoconstriction, speeding up the visceral circulation
4. Relaxes the sphincters.

If we consider symptoms on the basis of these physiologic reactions it will assist in determining the stage of the pathologic process. I do not mean to give the impression that this concept makes diagnosis a simple matter. Often these two syndromes overlap because the process has progressed to a point where it is both intramural and peritoneal, and the picture may be confused and obscured.

Dr. Sylvester will discuss the medical conditions under the title, "Surgical Simulations."

DR. PHILIP H. SYLVESTER.—My part is to discuss the situation as seen by the family pediatrician, or any family physician, or in consultation by a pediatrician and not by a surgeon.

What is really meant by "acute abdomen"? Some acute disturbance of the digestive function calls attention to the abdomen; vomiting, diarrhea, and acute sickness, constipation suggesting possible obstruction, pain referred to the abdomen by gesture or by direct statement, tenderness on pressure with or without spasm. Each of these factors is significant, more by the degree and the manner in which they interlock with one another than by any one alone.

Some years ago I investigated a thousand cases of "stomach-ache." In those patients who recovered, the final diagnosis was the one that seemed most reasonable. Cases with infection in the throat and neck only were over 40 per cent of the total; cases in which the chest alone was involved were 20-odd per cent; those in which the kidney alone was involved were over 10 per cent. Cases in which there was a true abdominal surgical condition were only 15 per cent.

Combined chest infection and a surgical abdominal condition were over 4 per cent. Throat and surgical abdominal condition totaled 4 per cent. Osteomyelitis cases simulating abdominal conditions totaled 2 per cent. I saw two patients with acute infantile paralysis who had been operated on for appendicitis.

There is a very distinct difference in a baby's reaction to intraperitoneal disturbances. Pain is tremendously significant. The terrific pain in volvulus and intussusception is practically diagnostic. In purely inflammatory peritoneal conditions, unless there is some mechanical involvement, there is not so much pain, tenderness, or spasm.

The importance of the bimanual rectal examination, the sense of resistance and the pain caused as your finger moves around, or as your fingers meet, is usually present in the truly surgical abdomen, even if you do not feel a mass.

In each of the cases, the following groups of signs and symptoms suggested an acute surgical abdominal condition, but further study showed they arose elsewhere or else arose within the abdomen but proved to be nonsurgical.

Throat, Neck and Mouth

- Tonsillitis
- Pharyngitis
  - a. Edema
  - b. Abscess
    - Lateral
    - Retropharyngeal
    - Peritonsillar
- Gingivitis (Trench Mouth)
- Parotitis
- Cervical Adenitis

Chest

External

- Trauma
- Fracture
- Osteomyelitis (ribs)
- Herpes zoster
- Hematoma—abscess
- Emphysema

Internal

- Asthma
- Pleuritis
- Pneumonia—not necessarily in the lower lobe
- Empyema
- Diaphragm—in pleurisy
- Poliomyelitis
- Trauma
- Mediastinitis
- Heart—
  - Endocarditis
  - Pericarditis
    with or without effusion
  - Congestive failure
- Cord tumor

Retroperitoneal

- Diaphragmatic hernia
- Pancreatitis, infectious (followed by glycosuria)
- Kidney
  - Trauma
  - "Pyelitis"
  - Stone
  - Ureters
  - Nephritis
  - Septic infarct
- Bladder
  - Distention—tumor (acute)
  - Cystitis

Abdomen—truly nonsurgical, but acute

- Wall
  - Abscess
  - Cord tumor
  - Hematoma, with abscess

Muscular lameness
Poliomyelitis
Trauma
Herpes zoster

Contents
"Adenitis"
"Aliergy"
Cardiac Liver
"Cholecystitis"
"Colic" and Colitis
"Enteritis"
Foreign Body
Infectious Jaundice
Mumps
Orchitis in abdominal testicle
Oophoritis
Post diphtheria
Purpura
Splenitis
Tuberculosis
Typhoid

We have all seen children who are sick with stomach-ache and tenderness. The next morning the child has a blistering red throat, and his stomach-ache and abdominal signs have disappeared.

Why would external trauma give a child stomach-ache? Why would it give him spasms unless it shook up something?

The first evidence of emphysema was very severe abdominal pain, related to the epigastrium with considerable involuntary spasm. The child had bronchopneumonia, which ruptured through and caused emphysema of the mediastinum with very acute illness for some time. Her emphysema was related to her stomach-ache which was nearly a surgical abdomen.

I do not know why the onset of acute asthma should give the appearance of an acute abdominal condition. I have seen in consultation cases of asthma with acute tenderness as well as rigidity that comes from the tremendous effort in breathing.

I do not know why stomach-ache and spasm should occur with intercostal or diaphragmatic involvement in poliomyelitis.

Three cases of cord tumor showed stomach-ache.

The clinical picture of a very acutely sick child with spasm and tenderness in the upper abdomen which gradually subsides and becomes evidently nonsurgical may be due to an acute pancreatitis. Two or three weeks later there was polyuria and crusting of the urine, and diabetes was found. I assume that was acute pancreatitis.

Kidney.—You will note nothing here but trauma and pyelitis. A small child fell downstairs, fifteen or twenty steps. He had a gray and green color, a slow pulse, and was cold. The blood pressure was low. There was a small mass in one side near the kidney with acute tenderness and spasm over the loin. On examination, the urine showed very few red cells. There was no fluid wave, no spreading of the tenderness. When the baby was warmed up and stimulated a little, his general condition improved and a diagnosis was made of an internally injured kidney without rupture of the capsule.

The next child who goes through the same accident will show increasing general symptoms but will not come back on stimulation and warming up. He will remain

in a condition of shock and will show evidence of acute blood loss. He will have no fluid wave and no increase in the area of tenderness and spasm. The autopsy will show retroperitoneal hemorrhage.

There *is* such a thing as pyelitis, though I always put mental quotation marks around the word whenever I think of it. Pyuria may mean a vast number of conditions, some congenital, some mechanical, and a great many infectious, relating to the ureter, the pelvis, and to the kidney substance itself. The most important condition under this heading is called the acute hematogenous kidney, meaning a general infection in which the kidney filters out and retains some of the organisms. They produce many or few small or large abscesses.

The beginning of the process is very acute and very severe. The child is prostrated, usually with signs of an acute surgical abdominal condition. Anywhere from twenty-four hours to five or six days later pus appears in the urine, and the child then, and then only, begins to get better.

A number of times a very acute pseudosurgical abdominal condition has been the mark of the onset of severe ordinary nephritis.

The bladder causes symptoms suggesting acute surgical abdomen under these conditions: distention, tumor, and actual inflammatory conditions of the bladder.

Contents of the Abdomen.—Until we can develop some sort of mechanical meter that will define in some adequate dimension the amount of pain, prostration, tenderness, and spasm, we have to depend upon the education of our fingers, our eyes, and our judgment.

Adenitis.—A child has symptoms of an acute surgical abdomen and the assumption is that he has an appendicitis, but on opening the abdomen, the surgeon finds a lot of small glands in the mesentery. Sometimes they may be thickly implanted in the meso-appendix. Another type will have a row of glands as large as walnuts near the base of the mesentery. It is difficult to learn what has been taking place.

Allergy.—If a child of known allergic tendencies has an acute abdominal condition and localized evidence of edema elsewhere, keep the surgeon away from him.

Infectious jaundice can produce errors in diagnosis if the patient is seen before the jaundice becomes apparent. If one will remember the slow pulse, even early, before the liver becomes large enough to be felt or before it is realized that it is the liver that is tender and not the peritoneum, one may avoid that pitfall.

Orchitis may occur in an abdominal testicle. I watched a patient three or four days before we decided there was nothing else, and the condition quieted down as orchitis from cases of mumps do.

Purpura becomes self-explanatory when the bloody flecks appear. The symptoms are not so definitely inflammatory as they are in other conditions.

Tuberculosis in all its forms may or may not be surgical, but by the time it reaches an apparent acute surgical manifestation, it is time to call in the surgeon and let him decide. I have seen a tuberculous adenitis produce a mass as large as an orange in the course of twenty-four hours' observation.

I am sure that children either do not or cannot localize abdominal pain or pain elsewhere. I am reasonably sure that the complex relations of the nervous system in childhood and possibly in late life have a great deal to do with it.

Pediatricians have to be responsible and see that their part is so well worked out as to leave no pitfalls for the surgeon. It is our duty to make a thorough study of each patient from the medical point of view to find the relative importance of each factor, to take carefully the history, and to make a careful evaluation of progress and symptoms and a conscientious and intelligent physical examination. Then and then only should we try to decide whether the abdominal symptoms we find point to a truly abdominal condition. In the real abdominal cases the progress of

symptoms or signs may be impressive, the patient's general condition may become worse and in particular, the abdominal symptoms may become more definite.

We have to determine these points early enough so that we may give the surgeon a chance to relieve a relatively well child rather than a desperately sick one.

CHAIRMAN COE.—When Dr. Sylvester says he hopes he is not trespassing, he brings out something which we all hope to see—that is, a mental attitude which will help us to realize, whether we are medical men or surgical men; that these conditions are parts of the same picture, instead of being two different pictures; that a child who has a surgical abdomen is a sick child and a child who has a medical abdomen is a sick child who should be treated as an individual and not as a collection of organs and symptoms; and that we should not, as soon as we have chased the disease out of the physical boundaries of our specialty, sit back and say, "Thank God, it is out of my territory! Now it is up to this other fellow!"

If we cooperate, the result will be better children's surgery and better children's care all the way through. But cooperation does not mean that one man makes all the diagnoses and then turns the cases over to someone else merely for the carpenter work. We must have surgeons who are pediatric-minded and who have a pediatric approach.

Now let us take up the other side of the picture. When is the acute abdomen really surgical? In other words, when does the pathologic process reach the point where we must use physical methods to restore normal physiologic function?

The symptoms which indicate that stage depend fundamentally, first, upon serious interruption of function, as for example in intussusception; second, on the presence of infection so severe that it overwhelms the physiologic defense mechanism, for instance, peritonitis; third, upon visceral trauma due to external force.

The first of these conditions due to serious interruption of function, in which the neuromuscular mechanism of the bowel tries to carry on normal function against a physical obstruction, will be taken up by Dr. Barbour of Louisville.

DR. PHILIP F. BARBOUR.—Dr. Sylvester emphasized one thing we all realize: surgeons feel competent to discuss signs and symptoms of any surgical condition in an adult, but they have a feeling of helplessness when it comes to children. This increases our responsibility because we have to do the thinking for ourselves and for the surgeon.

Obstructive lesions may be due to congenital maldevelopment in the gastrointestinal tract, such as malformations of the esophagus, pyloric stricture, atresia of the duodenum, jejunum, or ileum, embryonal faults in the course of the colon, undeveloped rectum, and the various defects at the anus. It is not necessary to comment upon these, except that stricture of the pylorus and pylorospasm are difficult to differentiate at times, as the pyloric stricture may not be complete. The tumor is certainly not always palpable. Vomiting at varying intervals, visible peristaltic waves, the dark green, so-called starvation stool, diminished output of a concentrated urine, and progressive loss of weight call for the surgeon if atropine and luminal with thick cereal feedings have failed to give relief.

Of the acute abdominal conditions, intussusception is fairly common especially in the earlier years. Starting most frequently at the ileocecal valve, it may often be palpated in different parts of the abdomen. Frank cases should be recognized early, the only time when operation offers much hope. An intussusception involving only a few inches of bowel is not so easy to recognize. Rhythmic pains accompanied by grunting and straining perhaps some vomiting without local tenderness in the abdomen (certainly not within the first twelve hours) and with the characteristic stools should make the diagnosis certain. These stools are often described as bloody

mucus, which may be confused with the bloody mucous stools of colitis and dysentery. The typical stool however is caused by the strangulation of the intussuscepted gut and is rather a blood-tinged serum which produces an extensive pinkish stain on the diaper, often with a clot of fibrin in the center. This blood-tinged stool may be evident only after enemas have been given. Occasionally resort to a barium enema with an x-ray picture which is quite characteristic will clinch the diagnosis.

We should remember that real pain will not be felt until the peritoneal covering has been involved, and this may arise only after many hours, when an operation will offer little hope.

Intussusception may be taken as a type of acute obstruction. I would like to discuss briefly certain physiologic and chemical results of the obstruction. Dr. Coe has explained the nervous mechanism that speeds up peristalsis in the presence of an obstruction; this is shown by the rhythmical contractures referred to before. Vomiting will remove the contents of the stomach and then of the bowel down to the obstruction. Such vomiting is often described as fecal because of the foul odor which is really due to the colon bacillus. Such material is highly toxic as is the brownish semibloody fluid which is collected at the obstruction. It is believed that the poisonous ingredient is histamine or some equally toxic amino acid. Certain changes take place in the blood also from obstruction or possibly from the vomiting—an increase in the N. P. N. and a decrease in the chloride ions. Some attach a diagnostic value to the diminution in these ions. At times an intravenous injection of 2 or 3 per cent sodium chloride will relieve the symptoms of an obstruction. There is opportunity here for profitable research work.

The vomiting not only removes the chlorine ions but causes dehydration. It is not necessary in this group to enlarge upon dehydration and the accompanying acidosis or alkalosis. Dehydration and toxicity are two pronounced dangers.

It is said that sloughing of the intussuscepted mass has occurred. Certainly such a fortunate ending is very uncommon. Otherwise, gangrene and/or peritonitis will be the mode of exitus if operation is long delayed.

Obstructions within the gut may result from old inspissated fecal matter particularly in Hirschsprung's disease or stricture of the rectum following ulceration, dysenteric, syphilitic, or tuberculous. Hair balls; worms, especially masses of ascarides; foreign bodies, metallic or otherwise; and colic from plumbism are other causes of obstruction. Gallstones, though almost never seen in children, cause an obstruction by irritation and consequent spasm of the wall of the gut; this may be true of other obstructive agents.

Volvulus is very uncommon. It is especially rare in childhood and would be very difficult to diagnose. Since the pain is more severe than in other types of obstruction, this might arouse suspicion of the condition.

Strangulated hernia is far more frequent in the adult because of the more strenuous life of the adult and also the inelasticity of the structures. Hernias of the intestine and/or stomach have entered the thoracic cavity through a defect of the diaphragm. They have been recognized by accurate physical diagnosis and by means of x-ray examination.

Meckel's diverticulum or other diverticula may be factors in causing intussusception. When inflamed, diverticula may lead to obstruction by kinking the bowel or in other ways. Bands, adhesions, and angulations may follow various infected and inflammatory lesions in the abdominal cavity.

Tumors of various kinds in the abdomen may exert sufficient pressure upon the bowels to result in occlusion. Nephromas, and hypernephromas, retroperitoneal tumors, dermoid cysts, teratomas, and we may include here diseases of the kidney or other organs which through nerve associations, as outlined by Dr. Coe, affect

peristalsis. Spinal cord lesions, as Pott's disease, tabes dorsalis, transverse myelitis and other involvements of the nerve centers are to be kept in mind. In any of these conditions the terminal stage may become acute and demand prompt attention although a proper history will elicit suggestive symptoms dating back for varying periods. We should add here that allergic pains are stressed by surgeons. They are certainly rarer in children than adults, and there will be other allergic signs which will help to corroborate the diagnosis.

A few moments must be given to the variation of symptomatology in the child as compared with that of the adult. Many surgeons rely upon the judgment of the pediatrician in evaluating the complaints of a child and put an additional responsibility upon us.

Pain is more variable and harder to localize in children. The pain of appendicitis may be located on the left side of the abdomen. If the obstruction is acute, the pain is sharper than in the more slowly developing cases. In general, pain above the umbilicus will arise from stomach or jejunum, while the pain from ileum or colon will be felt below the navel. Fear of the physician may exaggerate the sensation of pain and prove misleading, and on the other hand interest and attention will often distract the child's mind and real pain will be overlooked.

Vomiting is not always alarming as some children regurgitate food upon the slightest provocation. The stomach may have been completely emptied, and only retching remains to alarm one. If, however, there is a history of pain, nausea or vomiting, and constipation with or without rise of temperature and acceleration of the pulse, one should be on guard and avoid the age-accepted dose of castor oil. Enemas will be far better. If there is any question about the passage of flatus or fecal matter, the physician should superintend the enema himself and rely upon his own judgment as to the results.

As to the dangers, we pediatricians are fully cognizant of the importance of dehydration, acidosis or alkalosis, toxemia, high temperature and shock, in addition to the dangers peculiar to the various conditions that may arise. We shall be needed by the surgeon in the after-care of the child far more than is the case in surgery of the adult.

CHAIRMAN COE.—The next division of these acute abdominal conditions is one in which the primary reaction is the mobilization of leucocytes and the elevation of temperature and in which the sympathetic reactions are secondary. These conditions will be taken up by Dr. Sweet.

DR. CLIFFORD SWEET.—It has been emphasized that the child who has abdominal symptoms must be studied very thoroughly, even though the abdominal symptoms that appear to be alarmingly severe often clear up promptly without signs of any pathologic process within the abdominal cavity and are evidently caused by an acute process elsewhere, such as tonsillitis, pneumonia, or pyelitis.

A child may have any infection within the abdominal cavity to which adults are subject, but our principal concern is appendicitis and peritonitis secondary to appendicitis, or primary. The public must be educated to the fact that a child may have appendicitis at any age. Frequently, I operate upon a young child after the appendix has ruptured because the parents, thinking the child is too young to have appendicitis, attribute his abdominal pain to something he has eaten. We must also continue to impress upon our patients that the child who has abdominal pain or vomiting must under no circumstances be given a cathartic.

Pain is the one symptom which is always present when there is an inflammatory reaction within the abdomen. Ordinarily our attention is directed to the abdomen by this symptom and, even though the child may be inarticulate and usually inac-

curate as to the exact anatomic location of his pain, he commonly behaves in such a manner that his mother recognizes his discomfort and sends for his physician.

Fever may be absent; it is usually slight in the early stages and may remain in the lower ranges even when peritonitis is present. Many children have only from one-half to one degree. In order to convince the parents that I am making a correct diagnosis I usually have to explain that this slight rise in fever is usual. *Appendicitis, like diphtheria, begins with only a slight rise in body temperature.* When I see a child who has abdominal pain and a fever up to 103° or 103.5° or even 102° F., I search very thoroughly for something other than appendicitis.

Tenderness over the appendix region is usually present. However, when the patient is very young, its exact location and its degree may be difficult to ascertain. Also, if the appendix is located retrocecally, the tenderness may be slight and may be missed except when approached by means of a rectal examination. "Rebound tenderness" produced when the pressure of the palpating hand is suddenly released, more constantly reveals the presence of masked tenderness than does direct pressure.

Muscle spasm of the abdominal wall over an acutely inflamed appendix is usually present. When this sign is present, it points certainly to an inflammatory condition that probably needs surgical treatment. The variability of this sign in young children has been mentioned. It may be absent, and it is frequently intermittent in character. The intervals between periods of pain and muscle spasm when neither is constant may coincide, but one or the other may be absent while the other is present.

I once saw a child with evident acute appendicitis. His lower right rectus region was boardlike in its rigidity. The family asked for consultation before operation. When the surgeon examined the child's abdomen no muscle spasm was present, and I could see by the expression on his face that he was going to say, "There is nothing wrong with this child." Fortunately before speaking he again examined the child, and finding muscle spasm then present, concurred in my diagnosis. I removed a dangerously inflamed, swollen appendix.

All of these usual symptoms and signs which we have been discussing, namely pain, tenderness, and regional muscular rigidity, may be absent at one time and within a few minutes or hours be present so definitely that the diagnosis "acute appendicitis" is evident.

The examination by rectum, as has been pointed out, is extremely important in diagnosing what has happened inside of the abdominal cavity. However, if the rectal examination is not carefully made, it may yield incomplete or misleading information. The examining finger should be well lubricated with a good surgical lubricant rather than with vaseline and should be introduced through the anal canal slowly, advancing only during inspiration when resistance is at a minimum. If the patient has a small anus, it can be easily cocainized by mixing a few drops of 4 or 5 per cent cocaine solution with the lubricant, introducing some of the mixture well inside the anal canal with an applicator and then waiting a few minutes before introducing the finger. Even more important, the examining finger *should always* be introduced *first* into the left side of the patient's pelvis and bimanual palpitation should be done on this side before the finger is moved to the right side. The patient may object to the examination of his left side, but if he has an acute appendicitis or right-sided peritonitis, his evident increase in discomfort and the unmistakable change in the quality of his protests will seldom be misleading. The rectal examination carefully carried out, with the precaution I have mentioned, is the most useful method available in differentiating acute appendicitis from the other conditions mentioned.

The leucocyte count is not an infallible sign in acute appendicitis. Whether or not the child should be operated upon must depend upon the judgment of a

trained person after a careful study, not upon any single laboratory procedure. Some of the most acutely dangerous appendices I have removed have been accompanied by a normal or only slightly elevated leucocyte count. The recent studies on acute appendices made in Philadelphia* showed an elevated leucocyte count in only 76 per cent of proved cases of appendicitis at all stages. At times an elevation in the percentage of polymorphonuclear leucocytes, even though the total cell count is normal, will help to make up one's mind to operate. We must not depend upon the leucocyte count in acute appendicitis and disregard the other symptoms and signs which are telling us plainly to operate before it is too late to do so with the greatest safety.

When a diagnosis of acute appendicitis has been made, the child should be operated upon at once, unless some perfectly defensible reason for delay is present. I am not afraid of the after-results of an operation upon a child with acute appendicitis, but I am very much afraid of the after-results if he is not operated upon, both at the time and during the later years of his life.

I have little, if any, fear of adhesions in the abdominal cavity of a child who is properly operated upon. I have seen the abdominal cavity of a child who came to me with a ruptured appendix for which I could do nothing more than incise the abdominal wall and insert a drain. One month later he developed a secondary intra-abdominal abscess walled off by intestinal coils, for the relief of which I had to reenter the abdominal cavity and again insert drains. Three months later I did a prophylactic removal of the appendix and found only a single adhesion leading from the abdominal incision to the appendix.

When a child has symptoms and signs that cannot be explained by any reasonably probable diagnosis except appendicitis, he should be operated upon as soon as possible. Seldom does any one have a single attack of appendicitis unless his appendix is removed at the time of the first attack. A recurrent attack may come when conditions are very unfavorable for operation.

Many of my patients upon whom I operate early in an attack of appendicitis are able to go home from the hospital before the fifth day. After operation the child can be kept perfectly comfortable with sufficient doses of morphine, codeine, and sodium amytal, so that neither he nor his parents look back upon it as a "dreadful experience." The parents are spared the anxiety they otherwise wisely would feel that the child may have acute appendicitis when he was absent from them or from the region of the hospital and a competent surgeon.

When the attack of appendicitis is severe from the outset, when the history reveals previous attacks, when perhaps a cathartic has been given and one is reasonably certain the appendix has recently ruptured, operation should be delayed sufficiently to allow the inflamed area to be walled off. This is the one circumstance under which one needs an experienced consultant as well as a sixth sense to help him decide the best time for operation. Too early operation may cost the patient's life; too great delay may allow a secondary generalized peritonitis to develop.

Successful treatment of peritonitis secondary to ruptured appendix depends upon the treatment carried out both before and after operation.

The pediatrician must know the surgeon to whom he refers his patients, being certain that he gives a prominent place to the pediatric viewpoint among his other accomplishments. If one has reason to believe the child may have a ruptured appendix, with an abscess lateral to it, a right rectus incision may make it necessary to traverse and thereby soil healthy portions of the peritoneal cavity with consequent spreading peritonitis. The McBurney or Rockey-Davis incision enables the surgeon to work at the outer border of the inflamed area and establish drainage directly through the abdominal wall without traversing free peritoneal space.

*Acute Appendicitis in Philadelphia, A First, Second, Third, and Fourth Survey by John O. Brown, M.D.

After operation the patient should be kept in the Fowler position constantly. A child can be kept in this position only if the surgeon insists that he be supported with a full rise of the bed under his knees and a folded towel or sheet passed through the axillae and pinned to the top of the bed. If these precautions are not taken, the child will slump down often enough to permit spread of infection to the upper part of the peritoneal cavity. Pain must be prevented by sufficient doses of an opiate and rest secured with the addition of barbiturates when needed.

When obstruction or ileus is sufficient to cause intestinal contents to pass into the stomach, an indwelling Levin tube must be passed through the nose and the stomach kept clear by frequent flushing with warm water or normal salt solution and intermittent suction.

Dehydration must not be permitted. An abundant supply of water must enter the body by way of the rectum, subcutaneously, and intravenously. Glucose solution in from 5 to 10 per cent strengths must be added to this ingoing water to supply energy and prevent acidosis. Most important of all, the chloride level of the body must be maintained by giving a 1 or 2 per cent salt solution subcutaneously or in 2 or 3 per cent solutions intravenously. When distention of the intestine must be relieved, a 3 per cent salt solution intravenously is often the most effective means of emptying the bowel of its stagnant toxic contents.

CHAIRMAN COE.—I want to emphasize one thing Dr. Sweet has said; that is the matter of the Fowler position. A large amount of gas in the intestine may be swallowed. If the child is in the Fowler position, it is very hard for gas to go down through the duodenum and into the intestine. It usually comes up.

The third division of our subject deals with conditions due to trauma from external violence, and we shall discuss three of them: rupture of a hollow viscus, of a solid viscus, and visceral contusions. I shall not go into the matter of penetrating abdominal wounds, because the treatment is obvious in practically every instance.

The physiologic reactions on this group of conditions are rarely simple. Stimuli may come from external irritation or from mutiple injuries inside the abdomen. They may come from many different visceral segments, and because of their origin from many points our estimation of the condition on the basis of physiologic reactions must be very careful and very thorough. Many times the complete diagnosis is practically impossible.

The causative agent of abdominal contusions is important in diagnosis. There may be local force from a blow, a kick, a thrust with a sharp or blunt object, or a fall against something which gives comparatively localized impact; there may be diffuse force from crushing injuries or injuries distributed over a large portion of the abdomen; and finally there are injuries caused by falls.

The result of a localized impact is usually contusion or rupture of the underlying organ. Occasionally also such an injury causes a rupture of the mesentery.

As a result of diffuse force there may be rupture of any organ in the abdominal cavity. There may be rupture of the underlying organ at the place where the impact is greatest or rupture of a distant organ by transmitted force. Pathologic organs, as for example a hydronephrotic or tuberculous kidney, rupture more easily than normal ones.

The diffuse force of the impact may be transformed into a bursting force exerted by the contents of hollow viscera and may result in rupture of the intestines, bladder, stomach, or the great blood vessels, such injuries being remarkable for the unusual length of the tears.

Diffuse force also may cause a subserous rupture of any of the encapsulated organs or a rupture of the diaphragm. Traumatic diaphragmatic hernia is seen

with increasing frequence in recent years as a result of children being struck above the iliac crest by the bumpers of automobiles.

Falls on the feet cause hemorrhage from the liver or from mesenteric vessels because of the sudden strain on the suspensory ligament or the root of the mesentery. Falls on the abdomen, side, or back result in the same lesions as those due to diffuse force.

The initial symptoms of abdominal trauma are usually those of the general systemic reaction to shock—pallor, sweating, shallow respiration, small rapid regular pulse, low blood pressure, restlessness, and nausea. Localizing symptoms develop later as the general picture of shock disappears.

Rupture of the intestine gives rise to the sympathetic stimulatory syndrome. Peristalsis is reduced; capillary vasoconstriction causes dilation of the great veins with resulting pallor and small pulse; and the raising of the sensitivity threshold slows reflexes so that there is little if any rigidity and pain. There would be distention but this is prevented by the escape of intestinal contents through the rent in the bowel wall.

After a short period the somatic reflexes begin to appear in response to the increasing irritation of peritoneal surfaces by intestinal contents, and pain and rigidity, both local and general begin to appear. There is increasing tenderness, and respiration becomes thoracic because the child does not want to put pressure on his sore abdomen. Later the general reaction to infection develops, and there is fever, leucocytosis, etc.

Rupture of the bladder may be either extraperitoneal or into the peritoneal cavity. If extraperitoneal, the sympathetic reaction is minimal, and the somatic reaction is prominent. There is pain, local tenderness, rigidity, tenesmus, and there is blood in the urine if the opening in the bladder is not so large that the child cannot pass urine.

If the rupture is into the peritoneal cavity, the symptoms are those of sympathetic stimulation but are not marked at first and develop only slowly because the urine of children is usually not infected, and the symptoms are due to a rather mild chemical stimulation.

Rupture of a solid viscus may be subcapsular or complete. If subcapsular, the sympathetic inhibitory syndrome is early and marked, increasing with the increase of pressure beneath the capsule. Peristalsis is stimulated; sphincters are relaxed; and the sensitivity threshold is lowered. With the lowering of this threshold the somatic reaction appears quickly, and there is marked local pain, tenderness, and rigidity—the protective reflexes. If the rupture is complete, the inhibitory syndrome is minimal or may be absent, and the stimulatory syndrome develops very gradually on account of the fact that blood is nonirritating to the peritoneal surfaces. As a consequence, there may be few symptoms, and we are lulled into a sense of false security only to find general systemic signs of hemorrhage unexpectedly and suddenly developing. The child who complains very little after a possible injury to a solid viscus is the child to be watched most closely. I feel that such a child should be hospitalized and under constant observation with a half-hourly record of pulse and blood pressure.

If hemorrhage is intramesenteric, it causes the sympathetic inhibitory syndrome because of the distention of the peritoneum. There is a marked reaction—peristaltic, circulatory, and somatic. Such a patient is acutely ill.

With retroperitoneal hemorrhage the signs are often extremely obscure. The child's tissues are soft. The areolar tissues are spread without much pressure on nerves. The child does not seem sick at first, and again we are in the presence of a condition which gives us a false sense of security, because the only evidence we may have of a serious condition may be the gradually developing signs of hemorrhage.

Visceral contusions, especially intestinal contusions, give a clear-cut picture of the sympathetic inhibitory syndrome. With an intestinal contusion there is an intramural collection of blood, serum, protein decomposition products, or even intestinal contents. The result of this is a marked inhibition of the brake mechanism with accelerated peristalsis and accentuation of the peristaltic rushes, a marked lowering of the sensitivity threshold with acute perception of pain and marked protective reflexes, and the vascular reactions of pallor, small pulse, sweating, etc. The very severity of the reaction in these cases is a reasonable indication that we are not dealing with one of the more serious conditions. A change to the reactions of the stimulatory syndrome is presumptive evidence that secondary necrosis and perforation has occurred at the site of the contusion.

The diagnosis of these conditions is usually beset with difficulties on account of the many confusing factors involved. The external injuries, such as superficial contusions, fractures, and hematomas, may present the major side of the picture at first sight. Shock is usually present to obscure the symptoms of the deeper injuries. There may be multiple intraabdominal lesions. Absence of distention, which might be expected, may be due to escape of gas into the general abdominal cavity through a rupture or perforation, in which connection let me suggest the use of the fluoroscope. It does not take as much time as an x-ray plate and is just as good in demonstrating the crescent of air beneath one of the domes of the diaphragm. The latent period which follows the initial recovery from shock is often misleading. It is the period for the most searching diagnostic study rather than for a relaxation with a feeling that recovery is beginning. It is also the period of choice for whatever emergency surgery may be necessary.

Shifting dullness may be a rather late sign and usually means that there is not less than one or two pints of fluid in the abdomen, for the demonstration of fluid in the presence of abdominal injuries is very different from the demonstration of ascites in a child whose abdomen is not acutely tender.

Surgery should not be attempted in the period of shock, unless it is absolutely necessary to stop the progress of a serious hemorrhage; then it is obligatory. We may not save the child, but if he is having progressive serious hemorrhage there is no alternative.

There may be abdominal symptoms from chest or spine injuries or injuries in some other part of the body. The location of the external trauma will often make one suspicious that the abdominal symptoms are purely secondary, and as soon as the period of shock has passed the abdominal symptoms disappear.

The history of the type and extent of the injury helps a bit, and the time elapsed is important. If we see the child several hours after the injury, the period of the initial symptoms has passed, and we must look for the symptoms which develop secondarily.

There are many other details which might be presented but we shall pass on to the general discussion, and let me remind you that personal experiences always add to the interest of a round table discussion.

DISCUSSION

DR. FREDERICK SCHLUTZ (CHICAGO).—One of the interesting things in my contact with surgeons has been the experience with renal stones and malformations. Those more often lead to errors in surgical diagnosis than anything else. A stone is probably missed as readily by the pediatrician as by the surgeon.

One case occurred in a child about eleven years old who had hematuria. A number of diagnoses had been made, none however of renal calculus. We had no difficulty in making a diagnosis of renal calculus from the x-ray examination. The case was particularly marked by obscure abdominal pain and the recurring hematuria.

I recall two rather interesting experiences with herniations in the abdomen and through the natural openings. One was a healthy infant about nine months old with vomiting and severe abdominal colic which developed rather suddenly. The condition had existed for some eighteen or twenty hours. One of the striking things was the peculiar type of fecal vomitus. I sensed an obstruction and by carefully searching found the tender area in the right femoral region and made the diagnosis of femoral hernia, incarcerated.

Another interesting case was a girl, eleven years old, brought in for abdominal pain. She had had this complaint for several years, off and on, always in the umbilical region. Since she had been relieved before by the usual remedies, dietetic and otherwise, the condition was not regarded as serious. She had practically no fever and only a very occasional vomiting but a peculiar tender spot in the umbilical region. The persistent location of pain led us to operate at that point, and an old incarcerated hernia in a subchronic inflammatory state was found.

One of the most difficult differential diagnoses to make is between slight intussusception that has existed forty-eight or sixty hours unrecognized and infectious diarrhea; the transverse intussusceptions especially are difficult to recognize. In both there is abdominal pain and quite a bit of vomiting. Intussusception does not always, even as late as forty or fifty or sixty hours, result in complete obstruction. I had one patient who went four weeks with remarkably slight symptoms. Finally, we made the diagnosis, and the surgeon found the intussuscepted gut.

Another puzzling thing is intussusception in the five- or six-year-old child. I have had four such cases due in every instance to a Meckel's diverticulum. The abdominal symptoms are rather puzzling, and the greatest reliance should be placed upon the amount of blood seen in the stool.

Another thing to mention is the extreme rapidity of spreading peritonitis in the young child.

I have encountered ruptured kidneys from trivial injuries in three cases. Once it was a kick in the back; another time it was a baseball bat; and in one case the child had fallen. I would not have suspected a rupture if it had not been for very sharp and marked hematuria that induced me to persuade a surgical colleague to operate and in each instance we found a ruptured kidney.

It does not take a great deal of trauma to rupture a viscus organ, at least in a child, as his back has very little muscular protection.

I have seen a suppurating ovary, ovaritis, in older girls. One was twelve years and one was thirteen. I made a diagnosis of appendicitis. One patient was not operated upon because the appendix was supposed to have ruptured; the child died. We discovered that it was not appendicitis but that the ovary was suppurating. Another case was diagnosed as appendicitis, but the patient was operated on and lived.

Dr. Sweet referred to abdominal adhesions, saying that they do not occur frequently in children. We had a case of a girl eleven years old who was brought in forty-eight hours before primarily because she had an abdominal complaint.

At nine months of age she had been operated upon for intussusception. At seven years she had a clean operation for appendicitis. The intussusception was not the type in which one would expect adhesions, but she had had for three or four years, a great deal of abdominal complaint called "indigestion." Her abdomen was quite distended. She had considerable vomiting and no fever. She was operated on and found to have a general peritonitis with ruptured bowel. I think adhesions probably were at fault.

I have twice seen appendicitis in which the appendix was filled with *Oxyuris vermicularis*. It was mildly inflamed but teeming with worms. One was an infant eighteen months old.

I once saw a roundworm cause an abscess of the liver.

I have not seen a hair ball myself, but a colleague had a patient, a girl ten years old, from whom he removed a hair ball about the size of a duck's egg. It caused some symptoms of obstruction and a good deal of abdominal complaint.

Duodenal ulcer is not as uncommon as we have thought. The children range from the ages of eleven to fourteen, and the duodenal ulcers have been proved without question.

We are too reluctant to examine by rectum. I learned long ago not to hesitate to make thorough rectal examinations and have been rewarded at times by finding interesting conditions that would have escaped me entirely without such an examination. I agree heartily with what was said about the type of rectal examination and how to make it.

Another thing that has been helpful in determining whether acute abdominal conditions are inflammatory and probably surgical has been the taking of mouth and rectal temperatures for comparison. There is sometimes a difference of two or three degrees. Such a difference is exceedingly significant.

DR. FRED P. WEBSTER (PORTLAND, ME.).—I have been particularly impressed with the responsibility of the pediatrician here. In the acute abdominal conditions we have more responsibility than in almost any other place in pediatrics, and the surgeon seems to rely more upon us. Where we need help most with the acute abdomen is in infancy.

The acute abdomen of a two-year-old child is quite different from that of the older child. The younger infant is always sick with an acute abdomen. The older child may not necessarily be very sick. It is well to remember that the infant will at the slightest abdominal disturbance hold the abdominal muscles rigid. We have no way of telling whether the abdomen is rigid or not. The infant breathes largely with his abdomen and this adds to our difficulty. If there is any trouble in the abdomen, it gives rise to apparent dyspnea. If we think the condition may be abdominal, I believe in calling on the surgeon for help. My errors in infancy have been in waiting too long.

Dr. Sylvester mentioned the tonsil or the nose and throat as simulating acute abdominal conditions. I wonder if during the nose and throat infections possibly the lymphoid tissue or some other tissue of the appendix does not take part in that same infection.

I would like to ask if it is the proper procedure to have the appendix removed in a case of measles where there are signs of appendicitis.

DR. S. R. KALISKI (SAN ANTONIO, TEXAS).—Giving barium by mouth and the administration of a barium enema, if there is any possibility of intussusception, will go far toward making an early diagnosis and assisting in differentiating those things requiring early surgical intervention from those things which can be watched.

I think my greatest mistake has been not in waiting too long to advise surgery and not in waiting too late for diagnosis but in acquiescing too rapidly or too frequently with a surgeon who has called me into consultation because he thinks a child should be operated on for appendicitis. I have lacked the courage to say "NO."

We should be guided by the age of the child with whom we are dealing. We can more readily afford to be conservative and wait when we are dealing with children two or four years of age and older than we can in the very young.

DR. LEE F. HILL (DES MOINES, IOWA).—I want to ask Dr. Sweet about the method of handling peritonitis in children. If you have a child you are quite certain has a ruptured appendix of perhaps twenty-four hours' duration, from the

general rigidity of the abdomen, and the child is in good shape, would you advise operating at that time or letting it go for a week or two until you are sure the process is localized?

Another question is if the child apparently has generalized peritonitis and is very ill, getting worse, with more and more distention in spite of anything you can do, and it looks as if the child is going to die, would you perform an enterostomy to relieve the distention and drain? Or would you let the child alone, taking your chances that he might possibly recover?

I am also interested in your opinion of the use of morphine in peritonitis.

I recall a child two years of age, who was brought in for suspected appendicitis. The child had slight fever, normal leucocyte count and definite localized tenderness. I started to make a rectal examination and found a roundworm, about an inch and a half long, sticking out of the rectum; we delivered it with forceps. The appendix was removed, and a roundworm was found stuck in the appendix, the full length of the appendix.

I wonder if giving a barium enema to a child with obstruction is always safe. A child, eight or ten years old, who for six months previous to the time of admission had a history of pain in the abdomen intermittently with blood in the stools. This time that she came in she was given a barium enema. Very shortly after that she developed definite evidences of intestinal obstruction with a mass that could be felt in the left side. It was obvious that the child had an intestinal obstruction, and at operation we found a definite intussusception. I will never forget the appearance of that child's intestine. There were chunks of barium all through the large intestine, and to get it out through the opening was a serious task. In intussusception when the barium enema does go by, it adds considerably to the danger at operation. Giving barium by mouth to a child when there is intussusception is a dangerous practice. Certainly a surgeon doesn't appreciate the possibility of having hard chunks of barium above the site of obstruction.

One should look upon the acute symptoms as appendicitis or an acute surgical condition, unless he can prove it something else. I have never regretted removing an appendix in a child, but I have regretted several times that I didn't remove it. I would much rather take out a normal appendix in a child, even if he had some other condition, because if done carefully appendectomy will not hurt the child much, and the appendix is out. But if it ruptures while you are waiting around discussing other things, you have a mess on your hands.

I quite agree with Dr. Sweet that the child with one attack of appendicitis is going to have another. It is much safer, if there is a reasonably founded suspicion of acute appendicitis in the child with localized tenderness, to urge surgical removal rather than to delay.

DR. JAY I. DURAND (SEATTLE, WASH.).—I was called to see a child with what seemed to me a perfectly typical appendicitis, so much so that I sent him to the hospital and called a pediatric surgeon. The child had some leucocytosis, some fever, intense rigidity and pain. When the surgeon operated, he found a normal looking appendix. The next day the child looked all right, but the day afterward he developed a swollen epididymis and testicle. Our urologist said that occasionally a supernumerary lobe of the epididymis becomes twisted and gives all these symptoms.

DR. F. F. TISDALL (TORONTO, ONTARIO).—I would like to ask Dr. Sweet his opinion regarding the use of enemas in cases in which we suspect there is appendicitis. That is, the small child with colic and sometimes a little vomiting and then diarrhea. Those cases can be cleared up so rapidly or helped so greatly by enemas. If there is appendicitis, do you think there is a great deal of contraindication?

DR. JOHN F. SANDER (LANSING, MICH.).—As I heard Dr. Durand and Dr. Sylvester talk about calling in their pediatric surgeons, it made me quite envious. We in the smaller towns do not have that opportunity. The entire responsibility devolves on our shoulders. A great many of you men are in the teaching profession. I should think, as a matter of pediatrics, it would be a benefit if young men could be encouraged to take up surgical pediatrics more often.

DR. JOHN RUHRÄH (BALTIMORE, MD.).—Dr. Osler said the difference between a general practitioner and a specialist was that the specialist made a rectal examination.

I would like to ask Dr. Sylvester how many of his 85 per cent of cases with abdominal pain afterward turned out to be appendicitis.

DR. SYLVESTER.—I imagine probably two or three, not more than three.

DR. RUHRÄH.—We found differential blood counts are of vastly more value than just ordinary total counts. The total count does not amount to anything unless you have a series of counts and the count rises. But a differential count, even one, may give a great deal of information as to whether the thing is mechanical or inflammatory, and two counts taken a few hours apart, or three counts, will give a tremendous amount of information.

CHAIRMAN COE.—May I ask, Dr. Ruhräh, whether you use the Schilling count?

DR. RUHRÄH.—No, we have been using plain counts.

DR. HARRY M. GREENWALD (BROOKLYN, N. Y.).—Several points Dr. Sylvester brought out are very important. For instance, if we encounter abdominal pain during an attack of mumps and attribute this to a possible orchitis, occasionally we may be dealing still with a gangrenous appendix. The same is true of tonsillitis and scarlet fever, and appendicitis not recognized will cause very serious consequences.

Dr. Sweet and Dr. Barbour touched upon the importance of a Meckel's diverticulum. One of the most frequent occurrences is ulceration with abdominal bleeding and pain. If this is unrecognized and the patient is allowed to go on, perforation may occur with a fatal fulminating peritonitis. This is a different type of peritonitis than that which follows perforation of a duodenal ulcer or gastric ulcer.

Diverticulitis may occur, and it is very difficult to differentiate this from appendicitis, except that abdominal distention and vomiting are likely to be more prominent symptoms in Meckel's diverticulitis than in appendicitis.

If a child presents intermittent abdominal pain and has rectal bleeding for which you can't account by the presence of fissures in the anus or ulcerations of the bowel or the colon and you are sure you are not dealing with a gastric ulcer or duodenal ulcer or purpura hemorrhagica, that patient should be operated on and a Meckel's diverticulum looked for. In that way you can avoid the possibility of perforation if an ulcer of a Meckel's diverticulum is present.

There is one other condition which produces the so-called syndrome of acute abdomen; that is ovarian cyst. I have seen a number of cases. The youngest was two years old, and the oldest, ten years old. When it is the left ovary, the diagnosis is fairly simple. A mass can be felt; usually the child complains bitterly of abdominal pain. The temperature is very rarely elevated above 100° or 101° F. at the most, and the child does not look as sick as he does when he has an attack of acute appendicitis.

When it is on the right side, however, the differential diagnosis from acute appendicitis is extremely difficult. This is a condition that should be kept in mind because it does occur and occurs with greater frequency than we are led to believe from reports in the literature.

DR. P. W. BEAVEN (ROCHESTER, N. Y.).—Dr. Sweet said when the temperature is very high and the leucocyte count is very high, he suspects that he is not dealing with appendicitis. Does he also find that when the pain is very severe, he probably is not dealing with at least an organic belly? A suggestion was once made that when a youngster has a pain so severe that he doubles up, there is generally no pathology within the abdomen. When he is lying in bed on his back, the suggestion was that the pain was pathologic within the abdomen.

I have been interested in a condition which hasn't been mentioned, a pain which is always localized at the navel, not to the right side. I reported about five years ago three youngsters in this category, and I have now had twelve who have had pain beginning at a perfectly definite time in life. This pain is always localized at the navel, never associated with tenderness or fever or other signs of appendicitis. After six months, sometimes after as long a period as two years, I have had the appendix removed. In all cases the pain has entirely ceased with the removal of the appendix. On examination of the appendix it looks like an hypertrophied tonsil with enlarged lymphatic follicle.

DR. GREENWALD.—I would like to ask Dr. Sweet if he thinks there is such a condition as chronic appendicitis in children.

DR. P. H. HERRON (SPOKANE, WASH.).—Quite frequently in diabetic acidosis we find a condition that simulates appendicitis. I have had two diabetic children with pain which lasted only during the acidosis. In both cases the pain was localized in the right lower quadrant.

DR. ALVAH L. NEWCOMB (CHICAGO, ILL.).—A child came in with symptoms over the whole abdomen, and the diagnosis was possible peritonitis. As soon as diabetes was recognized and the acidosis was relieved, the child recovered.

I have seen four cases of appendicitis in diabetics, in three of which the appendix ruptured. Two of these were typical. Another was rather difficult to diagnose because the child had had scarlet fever, and had some scarlet fever antitoxin. About the tenth day there was some distention and tenderness and with it quite a bit of urticarial reaction; it was thought that this was probably an allergic affair. The rigidity became more severe, and the child was operated upon.

DR. J. W. ROSENFELD (PORTLAND, ORE.).—I would like to emphasize one point; that is, the more or less constant association of acute upper respiratory infection with abdominal pain. Do you regard these low-grade affairs of pain and resistance which are more or less transitory and which clear up with the subsidence of the upper respiratory infection as real appendix involvements? I find that the vast majority clear up without any permanent injury. In a few cases that go on to appendicitis requiring operative interference, it is rare that I cannot determine an acute upper respiratory involvement of some nature.

DR. PHILIP ROSENBLUM (CHICAGO, ILL.).—In pediatrics the history is probably more important than in adult specialties. If we go into the history, we will get a lot of information before we examine the child. When a careful history suggests an intraabdominal condition, before making an examination, watch the reaction of the baby being held snugly against the mother's chest. If the baby cries, look out.

A small percentage of patients with intussusception have no blood in the stool.

Once or twice a year cases of pericarditis with abdominal pain are admitted; the appendix is removed; the temperature persists; and later the pericarditis is found.

DR. E. C. MITCHELL (MEMPHIS, TENN.).—I have had two experiences which may be of interest. The first child was operated on for double mastoid. The following morning the child developed acute abdominal pain. He naturally had a high leucocyte count. Physical examination showed pain and tenderness. We were in a quandary, but we operated and found a very acute appendix on the verge of rupturing.

The second patient, a child of about nine years, was brought in with an acute obstruction, temperature of 105° F., vomiting, almost semicomatose with an enormous abdomen. The history was that this child had had obstipation since birth and had had several previous obstructions, which had been relieved by high enemas. This time we were unable to give anything at all, even gas, by a high enema. So we performed a colostomy, and a large amount of feces, very thin in character, spurted out. A diagnosis of acute obstruction on top of Hirschsprung's disease was made, and the child recovered in a few days.

As soon as the condition warranted, we injected a large amount of barium and found an enormous colon, typical of Hirschsprung's disease. After waiting a day or two, we found the lower segment of the bowel did not empty itself of the barium at all. The x-ray pictures showed nearly all to be present except what had gone out of the artificial opening. We then gave a spinal anesthesia, and the bowel was promptly emptied. The child was sent home. After a short time, he was brought back because the artificial opening did not close. We again gave spinal anesthesia, cleared out what was left in the lower bowel, and the neurologic surgeon performed a sympathectomy. The lower bowel then began to take on its function, and in a short time the artificial anus closed. Now, three years later, the child is in perfect health.

DR. SYLVESTER.—In the case of mumps that worried us so much, the abdominal signs and undescended testicle were on the left side.

Dr. Greenwald spoke of appendicitis occurring in infectious diseases. A child was sent in with very acute abdominal symptoms, as a case of peritonitis. He had Koplik's spots. It was decided it was probably the beginning of measles and bronchopneumonia. Measles developed; pneumonia developed, but the abdominal signs persisted. The lesions cleared up; the pneumonia subsided, but the abdominal signs persisted. The surgeons finally operated and found pneumococcus pus.

Our opinion should be based on a carefully made physical examination and a carefully reviewed and intelligent interpretation of what we find. Then only do I think we have a right to think seriously of surgery, but we must think of surgery and get our minds made up early enough to give the child a chance.

A child called me up one night in the absence of her parents and said, "Come out here, Dr. Sylvester, quick. I have my pericarditis all over again." She had a very serious attack before. I went out and found a very acute right-sided pain with spasm. I went again the next morning and her eyes were yellow; her liver was down; and her urine was yellow.

I have learned that to make a diagnosis of appendicitis in a girl without examining the urine is to run into a great deal of trouble. Absence of pus does not necessarily mean absence of something wrong with the kidney, but if you have a pretty definitely localized appendix group of symptoms and find pus in the urine, I think you are justified in waiting until things go a little more definitely one way or the other. Still, I have seen the two together.

A white blood count of 15,000 in a child of five with a change of 3,000 is no more significant than technical error. I want to ask Dr. Sweet if this young man who had a case of peritonitis unoperated upon had been under observation long enough to be pretty sure there were no signs of adhesions.

Dr. Barbour spoke about subsequent attacks. All of us agree, I think, that when a child has had appendicitis definitely, the thing to do is take the appendix out as soon as you can.

I have a very dear friend who is an outstanding general surgeon. I have taken care of his children and have had surgical experience with three of them. The first was a boy with acute appendicitis. I had to drag him off to another surgeon and have him operated on. The second was a boy with "acute intestinal obstruction." We had to lock the father up and the surgeon he brought with him while a good nurse gave the boy enough enemas to relieve him of constipation. The doctor wanted to operate for obstruction. The third was a daughter. This will answer Dr. Webster's implied question about adhesions. I had her under my care from the minute she was born. They lived hardly a hundred yards from where I did, and I saw her unnecessarily a great many times, but she never had a stomachache that I knew of. When she was nine years old, I took her to the hospital and had another surgeon take out her appendix; in the appendix were two definite strictures from previous attacks.

I personally have had experience with three cases of normal appendices coming out of children with negative chest x-rays taken because they had symptoms which made us feel there was interference with the respiration from an abdominal cause. In each case the x-ray of the lungs was negative; a normal appendix came out; the child failed to get better, and at the end of a few hours a shadow was found which developed into pneumonia.

In relation to poliomyelitis and abdominal symptoms, I would like to mention a case of a child nine years old with a very typical infantile paralysis with hyperesthesia but without paralysis. She later developed right-sided paralysis of the legs.

Another child who was paralyzed in the right side came into the Children's Hospital for operation. The child developed fever, abdominal pain, vomiting, and everything associated with appendicitis except spasm. I found the child was very acutely tender in the appendix region and felt that since the child was paralyzed, he could not have spasm.

At operation there was found a green appendix. Dr. Sweet spoke of the diagnosis of appendicitis after rupture in a fifteen-month-old child. How do you make this diagnosis of appendicitis preoperatively?

I want to congratulate Dr. Schlutz on his attitude in Chicago of "allowing" the surgeons to operate. I think we pediatricians ought to take that very seriously. I do not "allow" them to operate. I ask them to see a patient when I think it may be a surgical case; they take the responsibility from then on.

We have a lot of lead colic in Boston because we have such rotten piping in the tenement part of the city. I don't know of any so severe as to simulate abdominal conditions. These patients don't have spasm or localized tenderness.

DR. BARBOUR.—The presence of pain in the abdomen and colic would make us think of an intestinal condition, and I think we should watch for the lead line along the gum to see whether lead is a factor in the case.

A valuable sign of appendicitis is the pain on release of pressure over the abdomen. Muscle guard is not so well developed in children and is not so easily elicited, but release pressure will be quite helpful in diagnosis. Rectal examination may be necessary and is quite a help in otherwise doubtful cases. The blood count is

also quite an aid. The counts will not be high until the peritoneal coat has become invaded, and then a high polymorphonuclear count will be convincing, especially if the young and stab forms are very greatly increased.

I have seen both transient and partial intussusception correct themselves, but one certainly feels much safer when the abdomen has been opened by a competent man.

I have seen perforating ulcers of the stomach, duodenum, and ileum usually resulting in death. We should not exclude them on account of age.

In reference to Dr. Coe's discussion of rupture of an internal viscus from trauma —air free in the abdominal cavity will cause a tympanitic note over the liver; the upper level of tympany will probably be the third or fourth rib.

I am glad that Dr. Sylvester gave so complete a list of the extraabdominal diseases which might present baffling symptoms in the abdomen. Of these, pneumonia and localized pleurisy can easily be very deceptive. The association of infections of the tonsils and the lymphoid tissue of the appendix, rheumatic myositis accompanying scarlet fever, and the other lesions mentioned by him should put us on guard, for the general surgeon, as a rule, is apt to disregard such points.

Dr. Sweet has emphasized the importance of early diagnosis and operation in cases of appendicitis in the young. In the adult there seems to be a mechanism which will wall off a perforated or gangrenous appendix. This is absent in childhood, and the necessity for prompt intervention in children is therefore paramount. The Schilling count is of value and also the pain on release of pressure, but the pediatrician needs a sixth sense of intuition, if he will save all cases of acute appendicitis in the very young.

Someone spoke about the absence of blood in intussusception. I have never failed to find blood in intussusception, not always from the voluntary movements, but when saline is used to wash out the bowel, there will be some pink tinge to it. Perhaps not sufficient evidence will come out of the rectum in the ordinary way.

In one case I used the barium enema, and when it came out, there was a pink tinge on the white that was quite evident. I imagine there are a great many mild cases of intussusception that probably reduce themselves, never come to operation, never become significant. I have seen intussusception occur during an abdominal operation in a child while the surgeon was manipulating the gut.

Some one has said that it is always easy to diagnose intussusception. He is getting ready to have a disaster.

As to the matter of chronic appendicitis—I don't know whether children have chronic appendicitis, but I do know that children have repeated attacks of appendicitis. I have had a number of them operated on who showed that they had had previous attacks, mild in degree.

DR. SWEET.—Dr. Ruhräh's observation about the making of several blood counts is not one to be depended upon. I recall one girl in whom I had four blood counts in the course of twelve or fifteen hours. I couldn't quite make up my mind to operate on her. She had a retrocecal appendix 50 per cent gangrenous when it was taken out. Her pain was the one symptom that made it apparent she should be operated on. The white count never rose above 10,000, and the differential count was not enlightening.

Dr. Beaven's observation about some of the children who have continual abdominal pain—it is a symptom the mother consults you for constantly, until finally you are more or less driven to take out the appendix. These children are completely relieved when the appendix is taken out. It seems to me they are very generally the type of children who have a good deal of lymphoid tissue and very often are

frankly allergic. The best example I have of that is a boy whom I have followed from the time he was a baby until he was sixteen years old. When his symptoms became so constant that he couldn't go to school because of pain in his abdomen, no fever, no leucocytosis, I removed from his abdomen a very large appendix, one that was the size of my finger, with a great deal of lymphoid tissue in it. The boy now, over a period of a year or so, has been entirely free from his pain. I have had a number of those children. Dr. Greenwald, I believe there is such a thing as chronic appendicitis, and I think probably this is an example.

It seems to me that of the whole gastrointestinal tract the appendix is simply the most vulnerable point and that it shares in any process which upsets the function of the whole tract.

Then there is the child who has acidosis with greatly distended abdomen, generally tender. One is in doubt as to whether or not there is an inflammatory process. If he has only acidosis, the abdominal distention will promptly go down after some intravenous glucose, and you can study his abdominal condition more easily.

Some one mentioned the importance of the history. There isn't any part of the study of a child to which I pay so much attention. When my internes present a case to me, I make it an invariable rule that they tell me what the history suggests before they tell me what they have found on physical examination.

Dr. Sylvester asked me about a child with a ruptured appendix whose peritoneal cavity I drained, then again had to go in for an abscess, and finally later took out the appendix to find he had only a single adhesion leading from the point where I introduced the drain to the appendix itself. The boy had been perfectly well for five or six years. Whether he now has any adhesions in his peritoneum, I don't know.

DR. SWEET.—When the patient is in the hospital, he is in good condition to be operated on, and the matter of the appendix and its dangers have been thoroughly discussed with the parents. Then is the time to take out the appendix. I am less fearful of the patient's recovery from removal of his appendix than I am from the removal of his tonsils. If a child is carefully operated on, has the proper kind of anesthesia, has the proper kind of postoperative care, enough opium in some form to relieve him from all pain, enough sedatives to make him sleep, he does very little vomiting, and he is usually quite able to go home early. So I think the time to take out his appendix is as soon as you have satisfied yourself that he has appendicitis and that you are not endangering him by operating in the presence of some other condition.

Dr. Sylvester's question as to whether or not I can diagnose acute unruptured appendicitis in a very young infant must be answered. I do not know. I have not done so, but intend to keep trying. I think the rectal examination will give the most reliable findings.

I think there are cases of chronic appendicitis as well as cases of abdominal allergy in which the appendix, being perhaps the most vulnerable point in the gastrointestinal system, produces the principal symptoms.

I have two ideas concerning acute appendicitis which I cannot prove. One is, that even early in an acute attack of appendicitis, considerable amounts of acetone appear more constantly in the urine than in the early stages of other acute infections. The other, brought to mind by Dr. Rosenfeld's remarks about abdominal pain in acute upper respiratory tract infections, is that the child who usually has abdominal pain as a prominent symptom with extraabdominal acute infections is more likely to develop appendicitis early in his life than is the child who has this symptom infrequently.

CHAIRMAN COE.—I have very little to say in closing this discussion. One thing which possibly someone else didn't mention but meant to is that 3 per cent sodium chloride intravenously should be used only when we are sure that there is no obstruction or when we are to operate immediately. Of course, that is perfectly self-evident, but I don't think it was mentioned. If you put in something to stimulate peristalsis, the obstruction piles up.

In peritonitis in which we have a ruptured appendix or a perforated ulcer that is continually feeding the peritonitis, if we can get in, very early operation is indicated. I have had particularly good success in reducing mortality by putting a tube clear in through a small incision to the opposite side of the peritoneum and washing out as much of the infected material as possible until the reflow comes clear, and then letting the good old peritoneum have a chance to do its work.

In hematogenous peritonitis, in which we do not have a constantly feeding opening from the intestine, it is much better not to operate. A certain percentage will die, but I think that the same percentage and the same ones would die if they were operated on. With the nonoperative treatment, many will get well.

Finally, Dr. Sanders, the condition you mention is very true. In a meeting of pediatricians we can all appreciate it much better than in a general surgical meeting. When we go to a general surgical meeting and note the lack of attention which is paid to pediatric surgery and some of the surgical monstrosities which are presented in papers having a bearing upon children by men who are otherwise good sugeons, we can see that the point you make is very well taken. The man who would do pediatric surgery must have delicacy in handling tissues, must be able to work through small incisions, must be pediatric minded in the preparation of his patient and in the after-treatment, and he must know the reactions of children. Again, he must be able to handle parents, which is often just as important as his knowledge of how to take care of the patient. All those things combined make a surgeon who is not the ordinary type, and constitute what we may term the nontechnical points of pediatric surgery.

I hope the time will soon come when we shall have a much fuller teaching of pediatric surgery.

Erratum

In Volume 4, page 710, 1934, the remarks attributed to Dr. Charles N. Kennon were made by Dr. Warren Quillian, Coral Gables, Fla.

News and Notes

The following have been certified by the **American Board of Pediatrics** since the last report.

A. F. Abt, Chicago, Ill.
D. P. Arnold, Buffalo, N. Y.
M. H. Bass, New York, N. Y.
B. I. Beverly, Chicago, Ill.
R. A. Black, Chicago, Ill.
G. L. Bliss, Long Beach, Calif.
J. R. Boulware, Jr., Lakeland, Fla.
E. Boyd, Minneapolis, Minn.
C. E. Bradley, Tulsa, Okla.
A. Capper, Philadelphia, Pa.
P. G. Carson, Wichita, Kan.
L. Cohn, San Francisco, Calif.
G. L. Cook, Tampa, Fla.
E. P. Copeland, Washington, D. C.
A. B. Cowan, Fresno, Calif.
A. M. Dannenberg, Philadelphia, Pa.
B. S. Dunham, Toledo, O.
P. W. Emerson, Boston, Mass.
O. A. Faust, Albany, N. Y.
H. M. Gilkey, Kansas City, Mo.
J. H. Green, Waterbury, Conn.
David Greer, Houston, Texas
C. G. Grulee, Evanston, Ill.
B. T. Haessler, Milwaukee, Wis.
W. M. Happ, Los Angeles, Calif.
G. Heller, Englewood, N. J.
J. H. Hess, Chicago, Ill.
C. L. Holland, Fairmont, W. Va.
C. H. Hoover, Lancaster, Pa.
G. P. Hunt, Pittsfield, Mass.
M. Ingall, Boston, Mass.
G. L. Johnson, Jr., Englewood, N. J.
J. A. Jones, Philadelphia, Pa.
F. H. Lamb, Los Angeles, Calif.
E. Lewis, Washington, D. C.
C. S. Lipsitt, New Bedford, Mass.
W. London, Perth Amboy, N. J.
W. D. Lyon, Akron, O.
S. D. Mills, Westfield, N. J.
A. G. Mitchell, Cincinnati, O.
F. T. Mitchell, Memphis, Tenn.
H. J. Morrison, Savannah, Ga.
H. T. Nesbit, Dallas, Texas
S. Newman, Danville, Va.
M. M. Nicholson, Washington, D. C.
I. Okin, Passaic, N. J.
James H. Park, Jr., Houston, Texas
C. C. Payne, Dayton, O.
H. H. Perlman, Philadelphia, Pa.
J. J. Pollack, Detroit, Mich.
N. A. Pokorny, Springfield, Mass.
R. E. Pray, Fargo, N. D.
R. E. Ramsay, Pasadena, Calif.
J. A. Ritter, Philadelphia, Pa.
J. H. Root, Waterbury, Conn.
E. Rosamond, Memphis, Tenn.
H. A. Rosenbaum, Chicago, Ill.
L. C. Rosenberg, Newark, N. J.
O. W. Rowe, Duluth, Minn.
H. F. Rowley, Rochester, N. Y.
H. Sherwin, Brooklyn, N. Y.
F. B. Smith, Baltimore, Md.
L. H. Smith, Portland, Ore.
R. W. Stahr, Fort Dodge, Iowa
M. Winters, Indianapolis, Ind.
D. Worthington, White Plains, N. Y.

The **American Board of Pediatrics** reports the following results of examinations held in 1934:

	TOTAL EXAMINED	PASSED	FAILED	PERCENTAGE OF FAILURE
Group I	10	7	3	30
Group II	19	15	4	21
Group III	24	17	7	29
Total	53	39	14	26.4

Comments

OCTOBER and November of this year marked the full development of the Academy program when for the first time regional meetings were held by all four regions. The October programs in Regions I, III, and IV were chiefly clinical sessions at which a large number of papers were presented. Region II held its meeting at the time of the meeting of the Southern Medical Association in November. Approximately 350 were in attendance at the Region I meeting in New York and 150 at the Region III meeting in Minneapolis. Between the annual meeting of the Academy with its round table and panel discussions and the clinical regional meetings, something of interest and value can be found to satisfy the needs and desires of every one.

What might be called the organization period of the Academy is over. We have a large interested and enthusiastic membership. Those who were skeptical of its need and desirability are now convinced. The scope and character of the annual and regional meetings are defined, and the attendance this past year shows the interest of the membership. The Journal, which it launched in the midst of an economic depression, is now firmly on its feet. The Academy has united over seven hundred pediatricians and has the potential power of becoming the most important influence in the medical care of children in the United States.

The future must be carefully considered, or else this potential power will gradually waste away or may even through unwise leadership be directed into channels that are not worth while. We have, for example, a number of organizations interested in the economic phases of medicine. This is, in our opinion, a field for the American Medical Association, and not one for an organization, such as the Academy, which represents a restricted medical field. It is an Academy of Pediatrics, and our field is that of pediatrics. There is no question but that this field at the present time, and for many years to come, is large enough for all the activities which the Academy can undertake. Worth-while activities which come to mind are legion. Groups or reference committees interested in medical education, public health education, parental education, physical education, and the like can do much by the study of the present status of these fields and can offer much needed medical guidance in their development and work. In the field of clinical and scientific medicine the opportunities and problems are unlimited. It has been suggested, for example, that a study and report by an Academy committee of such subjects as irradiated milk, pertussis vaccines, and scarlet fever toxins would do much to clarify the uncertainty which exists in regard to these subjects. An unbiased report by an Academy committee whose personnel is qualified to pass an opinion would be of much greater value than expressions by these same men as individuals. The time has come for the Academy to make use of its organization and take an active part in the field of pediatrics.

INDEX TO VOLUME 5

A

Abdomen, acute, in childhood, round table conference on, 857
Abscess, amebic, of liver, 750
Acidosis and alkalosis, round table conference on, 394
Aldrich, C. A., and Munns, George F., 590
Alkalosis and acidosis, round table conference on, 394
Allergy, round table conference on, 698
Amberg, Samuel, and Ghormley, R. K., 177
Amebiasis in children, 741
American Academy of Pediatrics, news, 219, 430, 569
  Proceedings, Annual Meeting 1934, 88, 112, 244, 394, 545, 698, 841, 857
Anemia, sickle-cell in a white American family, 609
Attitudes of and toward children, round table conference on, 112
Avertin, use of, in rheumatic encephalitis, 654

B

Barbour, Philip F., 857
Berman, J. K., 47
Beust, Theodore B., 841
Beverly, Bert I., 112
Black, Mervin H., Friedman E., and Esserman, A. L., 504
Blatt, Maurice, L., Herrold, R. D., Hoffman, S. J., and Schneider, M., 511
Blatz, W. E., 112
Blood findings, relation of, and "Correct" weight, to physician's estimates of nutrition of school children, 763
Bonar, Barnet E., 216
Bosworth, Olive, and Sutton, L. P., 61
Boyd, Carlisle S., and Lisa, J. R., 608
Boyd, Julian D., 841
Brown, Alan, and Summerfeldt, P., 727, 735
Bunch, C. C., 535
Bunting, Russell W., 841
Burgin, Leo B., and Higgins, H. L., 156

C

Carcinoma, primary, of the kidney in children, 608
Caries, dental, panel discussion, 841
Carotene, parenteral use of, in treatment of vitamin A deficiency in albino rats, 75
Catalepsy, occurrence of, in children, 330
Cavity, tension, in primary pulmonary tuberculosis in an infant, 51
Cervicovaginitis, gonococcus, lysed gonococci in treatment of, 511
Child, spastic, 431 (Book Review)
Cholecystitis, acute, with a gallstone in a child 11 years of age, 47
Cholelithiasis, acute cholecystitis with, in a child 11 years of age, 47
Chu, Fu-Tang, and Coady, H., 75
— and Murphy, A., 68
Clifford, Stewart H., 139
Clinics, child guidance, quarter century of development, 288 (Book Review)
  pediatric, University of Minnesota, 365
  University of Minnesota at Rochester and Pediatric Section at Mayo Clinic, 83
  Western Reserve University, 823
Coady, Harriet, and Chu, Fu-Tang, 75
Coe, Herbert E., 370, 857
Cold, common, and allied upper respiratory infection, 573
Comments, 138, 289, 432, 571, 726, 882
Committee on hospitals and dispensaries, 274, 422
Cooke, Jean V., and Mack, J. K., 601
Cowie, D. Murray, 244
Critical review, methods of testing hearing in infants and young children, 535
  neurologic conditions in infants and children, 830
  problems of newborn, 216
  surgical pediatrics, 370
  tuberculosis in children, 680
Croup, acute infectious, 433, 617, 776
Cullen, Glen E., 841
Cyst, congenital, of larynx, 491

D

Davis, Clara M., 573
Development, physical, standards of, for reference in clinical appraisement, 194
Digitalis manifestion, auricular fibrillation as early toxic, 811
Dohan, F. Curtis, 756
Dystrophy, pseudohypertrophic muscular, effects of glycine in, 359

E

Edelman, S. D., and Haber, G. B., 520
Eley, R. Cannon, 830
Encephalitis, rheumatic, (chorea insaniens), 654

Endocrinology in infants and children, 659
Enema, fluoroscopically controlled, in reduction of intussusception, 819
Engle, Earl T., and Smith, P. E., 163
Ephedrine sulphate in treatment of bulbar type of poliomyelitis, 29
Esserman, A. L., Friedman, E., and Black, M. H., 504
Exanthem subitum with encephalitic onset, 800

F

Fibrillation, auricular, as early toxic digitalis manifestation, 811
Fischel, Marguerite K., 431 (Book Review)
Fischer, Vincent E., 491
Flutter, auricular, in newborn infant, 802
Friedman, Emanuel, Esserman, A. L., and Black, M. H., 504
Frisch, Irving A., 654

G

Gangrene of extremity in newborn infant, 756
Ghormley, Ralph K., and Amberg, S., 177
Gibson, Stanley, 545
Glycine, effects of, in pseudohypertrophic muscular dystrophy, 359
Goldberg, Samuel L., and Rambar, A. C., 513
Gonococci, lysed, in treatment of gonococcus cervicovaginitis, 511
Goodwin, T. Campbell, and Schwentker, F. F., 475
Gordon, Murray B., 659
Grayzel, H. G., Kramer, B., and Solomon, C. I., 299

H

Haber, G. B., and Edelman, S. D., 520
Halpern, L. J., 40
Hamilton, Bengt L. K., 394
Harper, Paul, 794
Hartmann, Alexis F., 394
Health program, pediatrician and rural child, 210
Hearing, methods of testing, in infants and in young children, critical review, 535
Heart disease, round table conference on, 545
Helmholz, Henry F., 83
Hernia, inguinal, in premature infants, 513
Herrold, Russell D., Blatt, M. L., Hoffman, S. J., and Schneider, M., 511
Higgins, Harold L., and Burgin, L. B., 156
Higgons, Reginald A., and Ostlund, E. O., 495
Hill, Lee Forrest, 680
Hoffman, Samuel J., Blatt, M. L., Herrold, R. D., and Schneider, M., 511
Hood, Marion, Nichamin, S. J., and Poncher, H. G., 741
Hormones, gonad-stimulating, from pituitary and from human urine, 163
Howe, Percy R., 841
Hoyne, Archibald, and Welford, N. T., 643
Huenekens, E. J., 244
Huntington, E. D., and Williams, R. E., 819
Hypoglycemia, chronic, in childhood, 299

I

Ichthyosis, congenital, 484
Imbecility, mongoloid, in the mongolian races, 352
Immunization, active, against pertussis, 590
scarlet fever by nasal route, 504
diphtheria, free, problem of, in school, 771
scarlet fever prevention by, 531
Infections, acute, round table conference on, 244
respiratory, upper allied, and common cold, 573
Insulin, use of, in nondiabetic children, 495
Intussusception, fluoroscopically controlled enema reduction of, 819

J

Jezer, Abraham, and Schwartz, S. P., 811
Jones, Sir Robert, life of, 431 (Book Review)

K

Kanner, Leo, 330
Kinney, F. E., and Sanes, S., 51
Kiser, Clyde V., and Stix, R. K., 763
Knox, J. H. M., Jr., 210
Koehne, Martha, 841
Kramer, Benjamin, Grayzel, H. G., and Solomon, C. I., 299

L

Landon, John Fitch, 1, 9, 16, 29, 33
Leucemia, lymphatic, resembling rheumatic fever in child, 61
Lisa, James R., and Boyd, C. S., 608

M

McCulloch, Hugh, 545
McQuarrie, Irvine, 365
Mack, J. Keller, and Cooke, J. V., 601
Mannerstedt, Gordon, 596
Meningitis, tuberculous, aspects of, and possibility of its prevention, 291
Mercury, ammoniated ointment, idiosyncrasy to, 794

Mettel, Howard B., 359
Mortality, premature infant, reduction of, 139
Munns, G. F., and Aldrich, C. A., 590
Murphy, Anna, and Chu, Fu-Tang, 68
Myocarditis, diphtheritic, 642

N

Neffson, A. Harry, and Wishik, S. M., 433, 617, 776
Newborn, loss of weight in, prevention of, by use of hydrating solution, 40
problems of, critical review, 216
tetany, low calcium, in, 341
News and notes, 136, 285, 570, 881
Neurologic conditions in infants and children, critical review, 830
Nichamin, Samuel J., Poncher, H. G., and Hood, M., 741

O

Osteomyelitis among children, 177
Ostlund, Elvira O., and Higgons, R. A., 495

P

Panel discussion, dental caries, 841
Parmalee, A. H., 112
Pertussis, hemophilus, endoantigen (Krueger), 590
in adults, 596
statistical analysis of cases of, 322
Peshkin, M. Murray, 698
Platou, E. S., 531
Poliomyelitis, acute, differential diagnosis of, 16
bulbar, ephedrine sulphate in treatment of, 29
preparalytic serum therapy in, 9
sporadic, 33
treated in Drinker respirator, 1
Polsky, Morris, and Schorer, E. H., 771
Poncher, Henry G., Nichamin, S. J., and Hood, M., 741
Price, H. T., 244
Pyuria, chronic, presacral sympathectomy, 735
persistent, 727

R

Race, mongolian, mongoloid imbecility in, 352
Rambar, Alvin C., and Goldberg, S. L., 513
Rat-bite fever, 520
Respirator, Drinker, poliomyelitis treated in, 1
Rettger, Leo F., 841
Rheumatic fever, lymphatic leucemia resembling, in a child, 61
Round table conference on acidosis and alkalosis, 394
acute abdomen in childhood, 857
acute infections, 244
allergy, 698
Round table conference on—Cont'd
attitudes of and toward children, 112
heart disease, 545
Rothstein, Jacob L., 341

S

Sanes, S., and Kinney, F. E., 51
Sauer, Louis W., 244
Scarlet fever, active immunization against, by nasal route, 504
prevention by immunization, 531
Schick, Béla, 698
Schless, Robert A., and Sherman, J., 802
Schneider, Maurice, Blatt, M. L., Herrold, R. D., and Hoffman, S. J., 511
Schorer, Edwin H., and Polsky, M., 771
Schwartz, Sidney P., and Jezer, A., 811
Schwentker, Francis F., and Goodwin, T. C., 475
Sherman, Jules, and Schless, R. A., 802
Smith, Geddes, and Stevenson, G. S., 288 (Book Review)
Smith, Philip E., and Engle, E. T., 163
Solomon, Charles I., Kramer, B., and Grayzel, H. G., 299
Stevenson, George S., and Smith, G., 288 (Book Review)
Stix, Regine K., and Kiser, C. V., 763
Streptococcus hemolyticus, occurrence of, in throats of children in hospital wards, 156
Stuart, Harold C., 194
Summerfeldt, Pearl, and Brown, A., 727, 735
Sutton, Lucy Porter, and Bosworth, O., 61
Sweet, Clifford, 857
Sweet, Lewis K., 352, 750
Sylvester, Philip H., 857
Sympathectomy, presacral for chronic pyuria, 735

T

Tetany, low calcium, in newborn, 341
Throat cultures, occurrence of streptococcus hemolyticus in, of children on hospital wards, 156
Tisdall, Frederick F., 841
Toomey, John A., 244, 322
Tuberculosis in children, critical review, 680
primary pulmonary, huge tension cavity in, in infant, 51
protective inoculation against, in infants by the use of heat-killed human tubercle bacilli, 475

V

Vitamin A deficiency, early signs of, in albino rats, 68
parenteral use of carotene in treatment of, in albino rats, 75

W

Wallfield, Mark J., 800

Wallgren, Arvid, 291

Watson, Frederick, 431

Weight, prevention of, initial loss of, in newborn, by use of hydrating solution, 40

Weight—Cont'd
"correct," relation of, and blood findings to physicians' estimates of nutrition of school children, 763

Welford, Norman T., and Hoyne, A., 642

Williams, R. E., and Huntington, E. D., 819

Williamson, G. Richards, 484

Wishik, Samuel M., and Neffson, A. H., 433, 617, 776

INDEX NUMBER

Vol. 5 DECEMBER, 1934 No. 6

Hospital Library

THE JOURNAL OF PEDIATRICS

A MONTHLY JOURNAL DEVOTED TO THE PROBLEMS AND DISEASES OF INFANCY AND CHILDHOOD

Official Organ for
THE AMERICAN ACADEMY OF PEDIATRICS

EDITORS

BORDEN S. VEEDER, St. Louis
HUGH McCULLOCH, St. Louis

EDITORIAL BOARD

KENNETH D. BLACKFAN, Boston
THOMAS B. COOLEY, Detroit
ROBERT A. STRONG, New Orleans
PHILIP VAN INGEN, New York
HAROLD K. FABER, San Francisco

Published by THE C. V. MOSBY COMPANY, 3523-25 Pine Blvd., Saint Louis, U. S. A.

Entered at the Post Office at St. Louis, Mo., as Second Class Matter

Copyright 1934 by The C. V. Mosby Company

Lest We Forget . . . Dextri-Maltose . . . . The Carbohydrate of Choice . . . . for over Twenty Years . . . Never Advertised to the Public

"The dextrin-maltose preparations possess certain advantages. When they are added to cow's milk mixtures, we have a combination of three forms of carbohydrates, lactose, dextrin and maltose, all having different reactions in the intestinal tract and different absorption rates. Because of the relatively slower conversion of dextrins to maltose and then to dextrose, fermentative processes are less likely to develop. Those preparations containing relatively more maltose are more laxative than those containing a higher percentage of dextrin (unless alkali salts such as potassium salts are added). It is common experience clinically that larger amounts of dextrin-maltose preparations may be fed as compared with the simple sugars. Obviously, when there is a lessened sugar tolerance such as occurs in many digestive disturbances, dextrin-maltose compounds may be used to advantage."
(Queries and Minor Notes, J.A.M.A., 88:266)

No. 1 Maltose 51%. Dextrins 42%. NaCl 2%. H_2O 5%.
No. 2 Maltose 52%. Dextrins 43%. H_2O 5%.
No. 3 Maltose 51%. Dextrins 41%. KCO^3 3%. H_2O 5%.
DMB Maltose 52.6%. Dextrins 39.8%. HO 1%.

Please enclose professional card when requesting samples of Mead Johnson products to cooperate in preventing their reaching unauthorized persons. Mead Johnson & Company, Evansville, Ind., U. S. A.

Why Dextri-Maltose Merits Professional Favor

Continued down from 1911

1933

"Of the double sugars, lactose would seem the logical one to use since it is the natural carbohydrate of milk; as a matter of fact, however, there seems to be no advantage in giving lactose. Pure lactose is expensive; most commercial preparations contain impurities which have a slight laxative action. Pure maltose is not used for infant feeding. Cane sugar (sucrose, saccharose) is under almost all circumstances an entirely satisfactory product for use with infants; it is inexpensive and is always available in pure form.

"There are times, however, when it seems advantageous to give part of the carbohydrate in the form of polysaccharide; this is the case when diarrhea is present or when large amounts of sugar are to be fed. Since time is required for the hydrolysis of the higher carbohydrate, the absorption of sugar is more gradual, and an alimentary glycosuria is less likely to develop. The amount of fermentable sugar present in the intestine at one time is limited; hence fermentation is less likely to take place. Another advantage claimed for the use of polysaccharides is that the osmotic pressure of these substances is considerably less than that of the sugar resulting from their hydrolysis; there is therefore less tendency for water to be drawn into the intestine and perhaps lost in the stool when large amounts of carbohydrate are fed in this form. Mixtures of dextrin and maltose are usually employed when a polysaccharide is desired. A number of preparations are on the market which differ somewhat in their properties. The liquid malt extracts should not be used in diarrhea; they contain impurities which are somewhat laxative, and are therefore sometimes used in the treatment of constipation."—*L. E. Holt, Jr. and R. McIntosh: Holt's Diseases of Infancy and Childhood, D. Appleton & Co., New York, 1933, p. 151.*

1933

In congenital hypertrophic pyloric stenosis, "The diet should be breast-milk whenever possible; if this is not available buttermilk or partially skimmed lactic acid milk to which dextri-maltose is added is the best substitute."—*L. G. Parsons and S. Barling: Diseases of Infancy and Childhood, Oxford University Press, New York, 1933, vol. 1, pp. 748-749.*

1933

In the treatment of diarrhea, "Omit carbohydrate from the mixture until the stools assume a putty-like consistency, rather dry, without odor or mucus. This usually does not require more than one or two days. After this time it is very essential that Dextri-Maltose be gradually added to the twenty-four-hour high-protein mixture. Should constipation follow, Dextri-Maltose No. 3 should be used instead of Dextri-Maltose No. 1. The addition of carbohydrate is usually accomplished by beginning with 1 level tablespoonful of Dextri-Maltose No. 1, and increasing gradually 1 tablespoonful every other day, until the baby is taking 5 or 6 tablespoonfuls of Dextri-Maltose in the twenty-four-hour feedings."—*M. A. Bridges: Dietetics for the Clinician, Lea & Febiger, Phila., 1933, p. 536.*

1933

In the treatment of diarrhea, "Carbohydrate must be added in a day or two, starting with two level tablespoonfuls of Dextri-Maltose to the day's formula and increasing to six or eight level tablespoonfuls as rapidly as the infant's clinical course permits."—*H. D. Lynch: The diarrhoeas of early life, J. Indiana M. A. 26:155-159, April 1933.*

1933

In the treatment of diarrhea, "Dextri-Maltose grs. 10, may be added to each feed for the first 48 hours, and this amount may be gradually increased. By the beginning of the fourth day the interval between feeds can be increased to three hours. The amount of the feed is then about 2 oz. with added dextri-maltose grs. 30, eight feeds being given in 24 hours."—*J. B. Young: Diarrhoea and vomiting in infants, Lancet, 1:677-680, April 1, 1933.*

1933

In the treatment of celiac disease, "A second point which is also of very great importance is that starch should, as far as possible, be given in the partially-digested form of dextrimaltose. In this form there is much less tendency to fermentative changes, the abdomen tends to be less distended and the stools less frothy."—*R. E. Steen: Coeliac disease, Brit. J. Child. Dis. 30:163-180, July-Sept. 1933.*

1934

In the postoperative care of pyloric stenosis, "If unable to obtain breast milk, a high protein diet is used; a formula of albumin milk and dextri-maltose is arranged to fill the caloric needs."—*E. G. Hummel: Congenital hypertrophic pyloric stenosis in infancy, J. Med. Soc. N. J. 31:152-156, March 1934.*

1934

"Boiled human milk was the food of choice, but if it was not available a cow's milk formula was substituted. For the past year only one formula has been used. It consists of equal parts of unsweetened evaporated milk and water with the addition of three per cent dextrimaltose No. 1. This formula gave excellent results and was used interchangeably with the boiled human milk with no unsatisfactory outcome."—*A. V. Stoesser and E. C. Perlman: The care of the premature infant, Minnesota Med. 17:70-76, Feb. 1934.*

1934

"The type formula used consists of simple dilutions of milk, water and some carbohydrate, usually dextrimaltose, although granulated sugar and lactose have also been prescribed."—*A. Tow: Simplified infant feeding, a four feeding schedule, Arch. Pediat. 51:49-54, Jan. 1934.*

1934

"Lactose has no special value when added to the feed, and many consider it to be less digestible than some of the cheaper sugars. When, however, we consider the case of the child who is prone to get indigestion, there is no doubt that in these conditions a mixture of dextrin and maltose is the most easily tolerated of all carbohydrates. . . . 'Dextri-maltose,' however, is tolerated when other carbohydrates produce fermented stools."—*D. Paterson and J. F. Smith: Modern Methods of Feeding in Infancy and Childhood. Constable & Co., Ltd., London, 4th ed. 1934, pp. 62-63.*

1934

For marasmus with intestinal intoxication, "Even in these small infants it is a very wise move to give nothing by mouth, but a 15% sugar solution consisting of 6 tablespoonfuls of Dextri Maltose No. 1 and 3 tablespoonfuls of cane sugar added to 1 pint of water. This should be offered to the infant every hour and it should be allowed to take as much as it will."

In marasmus without gastrointestinal symptoms, "The carbohydrates of choice are Dextri Maltose No. 1 or one of the starches."—*W. A. Hawk: A few of the commoner feeding problems of infancy, Univ. Toronto M. J. 11:218-229, May 1934.*

MEAD JOHNSON & COMPANY, Evansville, Indiana, U.S.A.

Please enclose professional card when requesting samples of Mead Johnson products to cooperate in preventing their reaching unauthorized persons

The Journal of Pediatrics

CONTENTS FOR DECEMBER, 1934

Original Communications

A Study of Forty Cases of Persistent Pyuria. Pearl Summerfeldt, M.B., and Alan Brown, M.D., Toronto, Ontario 727

Chronic Pyuria (Congenital Defect)—Presacral Sympathectomy. Pearl Summerfeldt, M.B., and Alan Brown, M.D., Toronto, Ontario 735

Amebiasis in Children. Samuel J. Nichamin, M.D., Henry G. Poncher, M.D., and Marion Hood, Chicago, Ill. 741

Amebic Abscess of the Liver. Lewis K. Sweet, M.D., Peiping, China 750

Gangrene of an Extremity in a Newborn Infant. F. Curtis Dohan, M.D., Merion, Pa. 756

Relation of "Correct" Weight and Blood Findings to Physicians' Estimates of Nutrition of School Children. Regine K. Stix, M.D., and Clyde V. Kiser, Ph.D., New York, N. Y. 763

The Problem of Free Diphtheria Immunization in the School. Edwin Henry Schorer, M.D., and Morris Polsky, M.D., Kansas City, Mo. 771

Acute Infectious Croup. Part III. Croup Associated With Specific Diseases. A. Harry Neffson, M.D., and Samuel M. Wishik, M.D., New York, N. Y. 776

Idiosyncrasy to Ammoniated Mercury Ointment. Paul Harper, M.D., New Haven, Conn. 794

Exanthem Subitum With Encephalitic Onset. Mark J. Wallfield, M.D., Brooklyn, N. Y. 800

Auricular Flutter in a Newborn Infant. Jules Sherman, M.D., and Robert A. Schless, M.D., Philadelphia, Pa. 802

Auricular Fibrillation as an Early Toxic Digitalis Manifestation. Abraham Jezer, M.D., and Sidney P. Schwartz, M.D., New York, N. Y. 811

Fluoroscopically Controlled Enema Reduction of Intussusception. E. D. Huntington, M.D., and R. E. Williams, M.D., Chicago, Ill. 819

Pediatric Clinics

The Babies and Childrens Hospital of Cleveland and the Department of Pediatrics, School of Medicine, Western Reserve University. Henry J. Gerstenberger, M.D., Cleveland, Ohio. 823

Critical Review

Neurologic Conditions in Infants and Children. R. Cannon Eley, M.D., Boston, Mass. 830

American Academy of Pediatrics

Proceedings: Fourth Annual Meeting of the American Academy of Pediatrics. Panel Discussion on Dental Caries 841

Proceedings: Fourth Annual Meeting of the American Academy of Pediatrics. Round Table Conference on the Acute Abdomen in Childhood 857

News and Notes

News and Notes 881

Comments

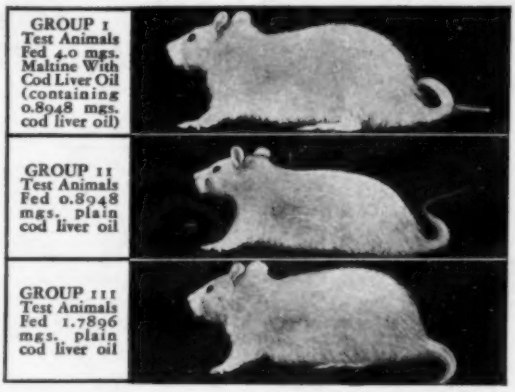
Comments 882

Index

Index 883

ENHANCING THE VITAMIN A VALUE OF COD LIVER OIL

IN ORDER to determine the comparative vitamin A value of the cod liver oil which forms 30% by volume of MALTINE WITH COD LIVER OIL, and the vitamin A value of plain cod liver oil, extensive experiments with animals have been conducted in the laboratory of one of the country's leading universities.



The results of these experiments confirm in every particular the conclusions drawn from previous tests, that the vitamin A value of cod liver oil as it exists in the MALTINE product is appreciably enhanced. The latest tests show that this enhancement amounts to a twofold increase.

Test animals in Group 1 were fed 4.0 mgs. of Maltine With Cod Liver Oil (containing 0.8948 mgs. of cod liver oil) and developed a highly satisfactory rate of growth. An equivalent amount of plain cod liver oil (0.8948 mgs.) fed to animals in Group 2 failed to produce satisfactory growth. It was not until twice this amount of plain cod liver oil —or 1.7896 mgs.—was fed to Group 3 that the growth rate approximated that of Group 1, in which half the quantity of oil was fed in the form of Maltine With Cod Liver Oil. It is evident from these tests that the vitamin A value of a given amount of cod liver oil as it exists in the Maltine product may be at least *twice* that afforded by the same amount of oil administered as plain cod liver oil.

Maltine With Cod Liver Oil is biologically standardized and *guaranteed* to contain vitamins A, B, D and G. Administered with either orange or tomato juice, vitamin C is provided. Biological report sent to physicians and institutions on request. Address The Maltine Company, 30 Vesey Street, New York, N. Y.

This Trade-mark Identifies the Only Genuine

Maltine

Reg. U. S. Pat. Off.

WITH COD LIVER OIL — Introduced in 1875

Member NRA
We do our part

MALTINE WITH COD LIVER OIL AND IRON IODIDE is also available. This is identical with MALTINE WITH COD LIVER OIL except that it contains two grains of freshly prepared iron iodide to each fluid ounce.